PESTICIDES ABSTRACTS

September 1976 - Vol. 9, No. 9

Abstracts 76-2102-2357

PSTAB 09 (09) 597-694 (1976)

Quarterly Index Issue

U.S. ENVIRONMENTAL PROTECTION AGENCY

U.S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF PESTICIDE PROGRAMS
TECHNICAL SERVICES DIVISION
Rm. EB-49, 401 M Street, S.W.
Washington, D.C. 20460

PESTICIDES ABSTRACTS (formerly Health Aspects of Pesticides Abstract Bulletin) is a monthly publication of the Technical Services Division of the Office of Pesticide Programs, Environmental Protection Agency.

The purpose of this publication is to foster current awareness of the major worldwide literature pertaining to the effects of pesticides as defined in the Table of Contents. The publication represents a monthly review of more than 2,000 primary and secondary sources. A list of periodicals from which articles have been abstracted appears annually in the January issue. Subjects and authors of abstracted articles are cross-referenced in quarterly and annual indexes. Reprint requests should be directed to the author of the complete article.

Publication of an abstract, annotation, or citation does not imply endorsement by the Environmental Protection Agency of the original article or of commercial materials, equipment, or processes mentioned therein.

CONTENTS

Volume 9 September 1976 No. 9

Page

General 597
(News items, letters, and editorials appearing in scientific and technical journals; information on production and use; and other items of general interest)

Monitoring and Residues 600
(Levels of pesticides in people or nonexperimental animals or in water, air, soil, food, feed, and other consumer products)

Epidemiology, Prevention and Treatment 617
(Frequency and distribution of pesticide poisoning in man or nonexperimental animals and treatment of clinical cases; morbidity and mortality; safety measures; decontamination; disposal of containers and wastes; legislation and regulations)

Toxicology and Pharmacology 624
(Toxicity studies in man and animals; effects on cells; organs, and systems; metabolic studies, biochemistry, interactions; antidotes and other treatment; mode of action. Ordinarily, articles dealing with efficacy studies are not included.)

Analysis 651
(Preparation of samples; analytical methods; instrumentation

Quarterly Index, July - September
Preface 655
Subject Index
I. Concepts 656
II. Compounds 668
Author Index
I. Personal 687
II. Corporate 694

76-2102. Tolle, A.; Heeschen, W. (Kiel, Germany). **Conclusions and recommendations resulting from the round table conference "Milk Hygiene."** *Arch. Lebensmittelhyg.* 27(2): 41-45; 1976. (24 references)

Twenty-six participants from 14 countries attended a World Association of Veterinary Food Hygienists Round Table Conference on Milk Hygiene. The general topics discussed included mastitis, machine milking and milk hygiene, pathogenic and saprophytic bacteria, biocides and environmental chemicals, and milk hygiene and public health. To minimize the transmission of organochlorine residues along the food chain and to keep milk relatively unaffected, organochlorine pesticides should not be used on crops, the lactating animals, or in barns and dairies. If total avoidance is not possible, correct dosage and cut-off times should be carefully adhered to. PCBs should never be used in open systems. The present situation should be monitored using representative samples of milk and feeds, and the results should be compared with the samples of milk and feeds, and the results should be compared with the national and international tolerances. The present situation carries no hazard for the consumer. More research should be done on the intake and output of highly toxic mercury compounds by the cow.

76-2103. Olkowski, W.; Olkowski, H.; Van den Bosch, R.; Hom, R. (Div. Biol. Control, Entomol. Dep., Univ. California - Berkeley, Albany, CA 94706). **Ecosystem management: a framework for urban pest control.** *BioScience* 26(6): 384-389; 1976. (23 references)

A pest management program was developed during 1970-1975 for Berkeley, California's 123 species of street trees. Citizen pressure and recognition of the shortcomings of pesticide use (resistance, target pest resurgence, secondary pest outbreaks, high cost) encouraged management personnel to try biological control techniques. Classical biological control has resulted in the control of 120 pest species in 253 importation projects throughout the world. This technique is ideally suited to urban insect management in that it represents low risk to humans and other animals, much of the vegetation in such areas is perennial, and the impact on the pest population is permanent. In addition, Berkeley estimated its savings at $22,500 per yr at 1970 prices. In 1970, parasitic Hymenoptera were imported from Europe against the linden aphid, and in 1972 parasite importations were made against the elm aphid and two aphid species of English oak. Because of the complexity of the theoretical ecosystem, the professional ecosystem manager must master a range of educational, rhetorical, sociological, psychological, biological, and physical scientific skills in addition to the horticultural and entomological ones. Such a professional might fulfill many parallel roles within other societal systems.

76-2104. Koehler, C. S. (Entomol./Plant Pathol. Coop. Extension, Univ. California, Berkeley, CA 94720). **Controlling insect behavior: theory and application.** *BioScience* 26(6): 399-400; 1976.

An international conference sponsored by the Rockefeller Foundation in April 1975 included major program sessions dealing with the sensory responses of insects to behavior-modifying chemicals, behavioral responses of phytophagous and hematophagous insects to host-produced chemicals and those produced by other insects, the nature and diversity of behavior-modifying chemicals in selected insect groups, and the status and prospects for behavior-modifying chemicals in pest management. The rapid progress made in recent years in the identification of behavior-modifying chemicals, especially pheromones, has exceeded the capacity of biologists and pest management specialists to develop useful applications for these compounds. Future research should concentrate on determining the behavioral and sensory-physiological mechanisms involved in insect reactions to behavior-modifying chemicals, the influence of the chemicals on target and nontarget organisms, and the short-range dispersal and long-range migration behavior of the target species. The more appropriate insect species to serve as candidates for suppression programs must be selected and behavior-modifying chemicals must be made operational.

76-2105. Stopford, W.; Goldwater, L. J. (Dep. Community Health Sci., Duke Univ. Med. Cent., Durham, NC 27710). **Methylmercury in the environment: a review of current understanding.** *Environ. Health Perspectives* 12: 115-118; 1975. (39 references)

The danger of methylmercury poisoning appears to be slight when the environment is not directly contaminated with methylmercury. Sediments rapidly bind mercury and decrease its availability in aquatic organisms, and they also have a greater propensity to demethylate than to methylate mercury. In noncontaminated aquatic ecosystems, the concentrations of methylmercury and inorganic mercury are many times lower than those that have been found to cause toxicity even in the most sensitive organisms. Methylmercury bound to protein is comparatively less toxic than methylmercury salts, and selenium present in this protein appears to be one of the major detoxifying agents for methylmercury. This is particularly important in seafood where there is an excess of selenium compared to methylmercury. Methylmercury poisoning has yet to be demonstrated in human populations not directly exposed to methylmercury or to food contaminated with it. Recent studies of populations who subsist mainly on seafood that is naturally high in methylmercury have failed to demonstrate any evidence of methylmercury poisoning.

76-2106. MacGregor, A. (Cent. Int. Studies, Massachusetts Inst. Technol., Cambridge, MA). **Analysis of control methods: mercury and cadmium pollution.** *Environ. Health Perspectives* 12: 137-148; 1975. (23 references)

Physical system conceptual models are developed to illustrate the various interconnecting pathways of heavy metal flow. The economic use of mercury and cadmium, as representative toxic heavy metals, is inventoried, and the

losses of each from the pathways of economic use are compared. Distinctions are made between high volume consumers and industries that are responsible for a large percent of total emissions or effluent loads; the burning of fossil fuels contributes twice as much mercury to the atmosphere as do emissions from industrial uses of mercury. The pathways of the metals in the environment are traced via the conceptual models, and a global mass balance is presented for mercury. The problem of high local concentrations vs. global metal flow is reviewed. Human health is most directly protected by monitoring of man's consumable resources; ambient air testing and testing of foods should be continued. Knowledge of past poisonings should be applied to the possible routes of exposure to heavy metal pollution. The best overall control system will be a mixture of several strategies, all aimed at reducing total metal flow: source controls, pathway control, safe product use, occupational hazard control, and human health protection.

76-2107. Brewerton, H. V. (Chem. Div. DSIR, Gracefield, New Zealand). **Pesticide residues in food and in the environment.** *N. Z. Dep. Sci. Ind. Res. Inform. Ser.* 97: 417-428; 1973. (50 references)

This paper reviews the changes which have occurred in the nature of pesticide chemicals used since the 1940s. In New Zealand agriculture, DDT and some other organochlorine insecticides came into considerable use in agriculture in the 1950s. DDT was mainly used to control subterranean pasture pests. To minimize the problem of DDT residues appearing in the fats of livestock fed on treated pastures, a number of restrictions were placed on DDT use on pastures during the early 1960s. During the 1950s the growth regulant herbicides such as 2,4-D and 2,4,5-T came into widespread use. For the most part, herbicides have not been observed to be significant polluters. The most notable unwanted side issue associated with herbicides was the finding that a dioxin impurity in the herbicide 2,4,5-T was extremely toxic and teratogenic. Fungicides are used to a lesser extent than either insecticides or herbicides. Some of the recently developed fungicides are quite stable in the environment, but they have not been observed as harmful in the broader aspects of environmental control.

76-2108. Grasblum, M. (Biol. Bundesanst. Land- und Forstwirtsch., 33 Braunschweig, Germany). **Aspekte der Wassergefaehrdung bei der Pflanzenschutzmittel-Ausbringung mit Luftfahrzeugen im Forst. [Aspects of water contamination resulting from pesticide application from airplanes over forests.]** *Schriftenr. Ver. Wasser-Boden-Lufthyg. Berlin-Dahlem* 46: 29-39; 1975. (7 references) (German)

Problems of the aerial application of herbicides and other pesticides over forest areas and of water contamination hazards are discussed on the basis of data on this mode of herbicide distribution in forests in West Germany. The total acreage treated with insecticides from airplanes has been steadily decreasing during the last five years, while the relative acreage treated with herbicides by this method shows an increase. Some 0.7-0.9% of the total forest area is treated with herbicides by the aerial method. About 90% of all areas treated are smaller than 5 ha. The preparations distributed from airplanes are usually dalapon, 2,4,5-T, possibly dichlobenil or chlorthiamid, maneb, zineb, mancozeb, endosulfan, trichlorfon, phosphamidon, dimethoate, lindane, propoxur, carbamates, and *Bacillus thuringiensis* preparations. According to the provisions of the regulations concerning water protection, 2,4,5-T, terbuthylazine, terbumethone, lindane, demeton-*S*-methyl, demeton-*S*-methylsulfoxide, Quassia extract, acridine bases, dicyclopentadiene, and *Bacillus thuringiensis* preparations are banned in forest areas from which the water flow time to the water capture area is less than 50 days. Pentachlorophenol, amitrole, and toxaphene are banned categorically in water capture areas.

76-2109. Klein, W.; Weisgerber, I.; Korte, F. (Gesellschaft fuer Strahlen- und Umweltforschung mbH, Inst. Oekol. Chem., D-5205 St. Augustin 1, Germany). **Chemische Aspekte der Umweltqualitaet. [Chemical aspects of environmental quality.]** *Staerke* 28(3): 101-106; 1976. (German)

General problems and observations are discussed on the impairment of environmental quality as a result of the ever-increasing production of a great variety of chemical substances. To correct any undesired effect of such chemicals on the environment as early as possible, it is essential to investigate representative model substances which are suitable for such studies in view of their production levels, areas of application, persistence, distribution pattern, degradability, and biological activity. Organochlorine pesticides, such as aldrin, DDT, dieldrin, and photodieldrin, are suitable for such model studies. Aldrin, dieldrin, and photodieldrin were found to be photolyzed in sunlight under natural conditions. The findings indicate the complete mineralization of these substances. The carbon dioxide production rate was determined to be 15% in 6 days.

76-2110. Rissanen, K.; Miettinen, J. K. (Author address not given). **Use of mercury compounds in agriculture and its implications.** *Tech. Rep. Ser. IAEA* 137: 3-34; 1972. (112 references)

This review of the agricultural use of mercury fungicides is concerned with their persistence in nature, their concentration into foods, and their effect on living things, including man. Specific topics included in the review are: the development and scale of seed treatment by organomercurials; the accumulation and effects of mercury fungicides on organisms (microorganisms, plants, animals, birds, mammals, and man); adsorption of mercury fungicides by soil and the natural Hg levels; decomposition reactions of mercury compounds (decomposition

by microbes, plants, birds and mammals); concentration by food-chains; and residues in victuals (both plant tissues and animals).

76-2111. Cremer, H. D. (Inst. Ernaehrungswissenschaft 1, Univ. Giessen, Giessen, Germany). **Pathogene Faktoren in Lebensmitteln pflanzlicher und tierischer Herkunft. [Pathogenic factors in foods of vegetable and animal origin.]** *Verh. Dtsch. Ges. Inn. Med.* 79: 46-54; 1973. (German)

General problems are discussed of pathogenic factors in foods of animal and vegetable origin. Such pathogenic factors include residues of pesticides, antibiotics and other drugs, toxic substances produced by microorganisms or from more or less harmless substances, natural substances present in animal and plant tissues, substances formed in the course of processing or culinary preparation, and food additives. The ban on DDT may be justified in developed countries but not in underdeveloped areas where it is indispensable in the control of malaria and other diseases.

76-2112. Barannyk, V. P.; Zhorov, V. O.; Kalashnykova, Yu. S. (Author address not given). **Rozvitok naukovihk doslidzhen' pro insektofungitsidi i galogenzamishcheni vuglevodni i zabrudnennya nimi navkolishn' ogo seredovishcha. [Growth of scientific experiments on insecto-fungicides and halogenated hydrocarbons and environmental pollution by them.]** *Visn. Akad. Nauk Ukr. RSR* 5: 87-91; 1974. (11 references) (Ukrainian)

A statistical study was performed on the amount and type of scientific literature published during 1963-1971 on the use of pesticides, their toxicity, and accumulation in flora, fauna, and sea organisms; also the future of pesticides in the atmosphere and oceans. The study, undertaken by the Hydraulic Engineering Institute of the Ukrainian Academy of Sciences, covered approximately 800 scientific reports published in USSR and world periodicals. It resulted in proper direction of research for the Institute—namely world ocean pollution by halogenated hydrocarbons. Benefits and damage due to pesticides exist in a precarious relationship. Intensity of research on the economics of pesticides, their future, and efforts against environmental pollution react sensitively to changes in science and production. As long as gross pesticide production increased and they were used widely in agriculture, industry, and medicine, literature on them concerned mainly their use, circulation in nature, and influences on ecosystems. As pesticide damage became more obvious, literature focused more on economics and control of pesticides. In general, the volume of scientific information increased eleven fold during the nine years studied; in certain areas thirty to fifty fold.

76-2113. Counc. Agric. Sci. Technol. (Dep. Agron., Iowa State Univ., Ames, IA 50010). **The phenoxy herbicides.** *Weed Sci.* 23(3): 253-263; 1975. (1 reference)

The phenoxy herbicides, 2,4-D, 2,4,5-T, MCPA, silvex, and related materials, are widely used as selective herbicides in crop production and the management of forests, ranges, and industrial, urban, and aquatic sites. The phenoxy herbicides are weak acids, only slightly soluble in water and petroleum oils. The acid is the active form; however, they are normally converted to water-soluble amines or oil-soluble esters for convenience in handling and application. The herbicides are absorbed by plant foliage, roots, and soft stem tissue. Herbicides are most useful when employed in coordination with other practices in vegetation management systems. The aim is to create conditions favorable to the crop, forest trees, range grasses, and other preferred species, and unfavorable to unwanted plants. Specific uses are discussed concerning croplands, pastures and ranges, forests, aquatic habitats, industrial lands, and urban lands. The toxicity of the phenoxy herbicides is known with reasonable accuracy on hundreds of species of plants and animals under a variety of conditions. Spray drift, toxicity, and residues are considered as problems.

76-2114. Schmidt, S. (4502 Bad Rothenfelde, Alte Salzstrasse 7, Germany). **Koennen wir unseren Koerper vor Umweltgiften schuetzen? [Are we able to protect ourselves from environmental pollution?]** *Z. Ther.* 13(7): 401-413; 1975. (German)

Inventory is given of the environmental pollution in soil, water, air, and foods, and possibilities are described of prophylaxis and detoxification by natural means. Pesticides, such as organochlorine preparations (aldrin, chlordane, dieldrin, endrin, heptachlor, malathion, lindane) interfere with enzymatic metabolism, and cause nerve damages. Most poisons are destroyed or detoxified in the organism by oxidation, reduction, hydrolysis, acetylation,and methylation, and are rendered water-soluble by glucuronic acids and sulfur-containing proteins. Metabolism can, however, also lead to more toxic metabolites, e.g., diethyl-*p*-nitrophenyl phosphate in the case of E 605 (parathion). Antidotes, cysteine, D-penicillamines, thiosulfates, enzyme preparations, ozone-oxygen mixtures, Polyerga, healing earths, enzyme-oxygen combinations, pollen diet, cell preparations, Korapur, red beets, yogurt, and Harpagosan tea can be used for detoxification, and also on a prophylactic basis. Biological and general natural methods of pest control should be preferred to chemical pesticides.

76-2115. Winnett, G.; Siewierski, M. (Dep. Entomol. Econ. Zool., New Jersery Agric. Exp. Stn., Rutgers Univ., State Univ. New Jersey, New Brunswick, NJ 08903). **Air sample studies for measurement of pesticide contamination from storage areas.** *Bull. Environ. Contam. Toxicol.* 14(6): 681-686; 1975. (3 references)

The possible escape of pesticide was studied from a building used for the storage of a wide variety of pesticides. Stanchions were placed near the exhaust fan of a room housing pesticide mixing apparatus, in various locations within the storage building, and at several points outside the building. The residues extracted from the stanchion screens were analyzed using flame photometric gas chromatography. The highest pesticide vapor concentrations were found within the air of the storage building; the concentrations of one of the stored pesticides, malathion, in the building ranged from 84.2 to 164.88 μg on the first day of sampling and from 82.52 to 92.25 μg on the seventh day. The relative concentration of malathion was inversely proportional to the distance from the storage building. A building situated between the storage building and stanchion 3 (placed outdoors) produced a barrier effect which resulted in consistently low pesticide concentrations at stanchion 3 (e.g., malathion measured 0 μg on day 1 and only 0.48 μg on day 7). A number of other organophosphate pesticides were also present in the air. Although it was impossible to calculate the actual pesticide concentration in the air, it is suspected that the pesticide levels within the storage building may have constituted a health and fire hazard. The use of stanchions is recommended as a means of detecting organophosphate pesticides in the air.

76-2116. Laska, A. L.; Bartell, C. K.; Laseter, J. L. (Dep. Biol. Sci., Univ. New Orleans, New Orleans, LA 70122). **Distribution of hexachlorobenzene and hexachlorobutadiene in water, soil, and selected aquatic organisms along the lower Mississippi River, Louisiana.** *Bull. Environ. Contam. Toxicol.* 15(5): 535-542; 1976. (9 references)

Samples of water, soil, and selected aquatic organisms taken at intervals along a 150-mile transect of the man-made levees of the Mississippi River (from Baton Rouge to Port Sulphur, Louisiana) were assayed for HCB and HCBD residues using gas chromatography. Both compounds, and particularly HCB, were found in soil samples from several sites; peak concentrations were found in the vicinity of large HCB-producing chemical plants. While concentrations of these compounds in the levee and ditch soil samples ranged from undetectable to nearly 900 ppb, the levels in most river water sediments were below 2 ppb. At least one of the compounds was detected in all water samples between Baton Rouge and New Orleans, one sample containing 90.3 ppb HCB and 1.4 ppb HCBD. Tissue residue levels in mosquitofish (*Gambusia affinis*) ranged from 71.8-379.8 ppb for HCB and from 112.8-827.3 ppb for HCBD. In crayfish, better correlations between tissue and ecosystem levels were obtained for HCB than for HCBD. The wide distribution of the compounds suggests various means of dispersal: accumulation in soil carried in suspension by river waters; runoff into levees which empty into the river; and contribution from air-borne sources.

76-2117. Benson, W. W.; Brock, D. W.; Gavica, J.; Loomis, M. (Idaho Epidemiol. Studies Project, Dep. Health Welfare, Boise, ID 83720). **Pesticide and mercury levels in pelicans in Idaho.** *Bull. Environ. Contam. Toxicol.* 15(5): 543-546; 1976. (10 references)

Twelve white pelicans (*Pelicanus erythrorhynchos*) collected in southern Idaho during early summer of 1974 were analyzed for tissue mercury levels using gas chromatography. The liver tissues contained the highest mercury levels (2.33-32.80 ppm), followed by the kidney (average 3.87 ppm), feathers (average 3.70 ppm), muscle (average 3.41 ppm), heart (average 2.37 ppm), brain (average 1.27 ppm), and bone (average 0.34 ppm). An additional bird, found dead, was examined for organochlorine residues. Total DDT levels ranged from 1498 ppm in the liver to 19 ppm in the brain, BHC ranged from 13.60 ppm in the liver to 0.635 ppm in the brain, heptachlor epoxide ranged from 13.20 ppm in the liver to 0.384 ppm in the brain, and dieldrin ranged from 203.00 ppm in the liver to 4.74 ppm in the brain. These pesticides may have been responsible for the bird's death. The mercury residues in the pelicans may have originated from mercury fungicide compounds used by farmers to treat their wheat seed.

76-2118. Adams, J. D.; Iwata, Y.; Gunther, F. A. (Dep. Entomol., Univ. California, Riverside, CA 92502). **Worker environment research. IV. The effect of dust derived from several soil types on the dissipation of parathion and paraoxon dislodgable residues on citrus foliage.** *Bull. Environ. Contam. Toxicol.* 15(5): 547-544; 1976. (18 references)

Slurries were prepared from a 20 ppm aqueous solution of parathion and dust samples representing six soil types. Slurry drops were spread on the top surfaces of living Valencia orange leaves and on Teflon sheets. Treated leaves removed at intervals up to 30 days after slurry application were assayed for parathion and paraoxon by gas chromatography. For all six soils, the initial parathion residue dissipation rates were linear, the rates then declining after about 10 days; the highest dissipation rate was observed with Laveen loamy sand. After 30 days, 4% of the original parathion remained on the Laveen soil, 15% on Santa Lucia, 20% on Windy, 8% on Madera, 14% on Visalia, and 2% on Pike's Peak clay. Dislodgable paraoxon residues first appeared on the second day, rose to a maximum by about the eighth day, and declined at a low thereafter. By the thirtieth day, paraoxon accounted for 17, 11, 5, 3, 15, and 34% of the applied parathion on the Laveen, Santa Lucia, Windy, Madera, Visalia, and Pike's Peak clay soils, respectively. Thus, soils reside on citrus leaves and influence the conversion of parathion to paraoxon.

76-2119. Cugurra, F.; Maura, G. (Univ. Inst. Pharmacol., Fac. Pharm., Genoa, Italy). **Mercury content in several species of marine fish.** *Bull. Environ. Contam. Toxicol.* 15(5): 568-573; 1976. (22 references)

The total mercury levels in samples of fresh, canned, frozen, and dried tuna, anchovies, sardines, mackerel, umbrina, cod, and oculate were determined by atomic absorption spectrophotometry. The mercury levels in the canned tuna varied from 0.274 to 1.326 ppm depending on the species and market brand. Among the other canned fish, the highest levels (0.416 ppm) were found in *Engraulis encrasicholus* caught in the Adriatic sea. The mercury levels in fresh *Oblata melamura* and *Clupea sardina* caught near Genoa were 2.593 and 0.484 ppm, respectively. Dried *Umbrina cirrhosa* and *Gadus morrhua* contained 0.707 and 0.326 ppm mercury, respectively. The continuous ingestion by humans of fish containing mercury in concentrations greater than 1-2 ppm is potentially dangerous, depending on the type of food consumed by a given population.

76-2120. Cockson, A.; Morgan, A. (Ind. Analysis Unit, Univ. Malawi, Limbe, Malawi). **DDT in babies' milk formula.** *Bull. Environ. Contam. Toxicol.* 15(5): 591-592; 1976. (4 references)

Samples of babies' milk formula powder purchased from a supermarket in Blantyre, Malawi, were extracted with distilled petroleum spirit, then with 5% diethyl ether in petroleum spirit. Clean-up was performed via extraction in situ using silica gel. Pesticide identification and quantitation were carried out by thin-layer and gas chromatography. No chlorinated hydrocarbon pesticides were detected in the 5% diethyl ether-petroleum spirit extract. Total DDT, consisting of similar quantities of *o,p'*-DDE, *p,p'*-DDE, *o,p'*-DDT, and *p,p'*-DDT, ranged from 0.10 to 0.14 ppm. These values are no higher than those reported for mothers' milk, indicating that babies fed formula milk are exposed to the same toxic hazards as breast-fed babies.

76-2121. Deubert, K. H.; Gray, R. S. (Univ. Massachusetts, Cranberry Exp. Stn., East Wareham, MA 02538). **Parathion residues in environmental samples from untreated areas.** *Bull. Environ. Contam. Toxicol.* 15(5): 613-616; 1976. (4 references)

Two soil samples and six oysters from the cranberry producing area of southeastern Massachusetts were assayed for parathion residues using thin-layer and gas chromatography. The soil was taken at a point 4 miles away from the nearest application site, and the oysters were taken at a point 2 miles from the nearest application site. Parathion residues in the soil were 1.3 ppb on SE-30 and 1.0 ppb on QF-1. The calculated p-values were 0.278 on SE-30 and 0.0313 on QF-1; the p-values for the shellfish were 0.286 on SE-30 and 0.293 on QF-1. The SE-30 data could not be reliably used for quantitation because an unidentified compound produced an unsymmetrical peak on SE-30 when used as described in this study; the unknown did not produce peaks on QF-1. The presence of parathion in samples from areas not treated with the pesticide suggests that parathion residues are translocated in the environment.

76-2122. Pollero, R.; Goyena, R.; Siquiroff, M.; Tumino, R. (Inst. Biol., La Plata, Argentina). **Removal of pesticides in water of Rio de la Plata.** *Bull. Environ. Contam. Toxicol.* 15(5): 617-622; 1976. (6 references)

Surface water samples from Argentina's Rio de la Plata were spiked with known amounts of heptachlor, malathion, endrin, and DDT, after which they were filtered through a membrane filter of pore size 0.2 microns or centrifuged at 10,000 × *g* for 20 min. Pesticide removal from the samples was determined using electron-capture gas chromatography. The removal of DDT and heptachlor from the filtered river water was greater than that of endrin and malathion; a similar phenomenon was observed with unfiltered water. Pesticide removal was attributed primarily to codistillation with water, not chemical decomposition or microbial metabolism. The chlorinated pesticides were immediately adsorbed by the suspended matter in the samples not subjected to filtration or centrifugation; the progressive removal of the pesticides in these samples may have been due to desorption and perhaps to decomposition. The sediment removed by centrifugation of pesticide-spiked natural water contained no malathion.

76-2123. Wood, G. W.; Stewart, D. K. R. (Agric. Canada Res. Station, Fredericton, New Brunswick, Canada). **Fenitrothion residues in blueberry fields after aerial forest spraying.** *Bull. Environ. Contam. Toxicol.* 15(5): 623-629; 1976. (2 references)

Surveys were carried out in 1972, 1973, and 1974 to determine the extent of fenitrothion drift into New Brunswick blueberry fields during spraying of nearby forest areas (3-8 km distant) for spruce budworm control. Residue levels on glass sampling plates were determined by flame-photometric gas chromatography. All but three of the fields located approximately 3 km from the sprayed forests contained fenitrothion residues ranging from 0.02 to 18.04 g/ha. In the 1973 survey, the fields located between 3 and 8 km from the sprayed forests also had detectable fenitrothion residues, whereas in the 1974 survey, detectable fenitrothion was found in only one field located more than 3 km from the sprayed area. Variability in the residue levels may have resulted from differing wind patterns at the time of spraying and from some aircraft coming closer to the blueberry fields than others. The biological significance of the findings is unknown because the effects of the observed fenitrothion levels on organisms affecting the blueberry crop are yet to be determined.

76-2124. Siddaramappa, R.; Sethunathan, N. (Lab. Soil Microbiol., Central Rice Res. Inst., Cuttack-6, Orissa,

India). **Volatilization of lindane from water in soil-free and flooded soil systems.** *J. Environ. Sci. Health* B11(2): 119-127; 1976. (10 references)

A detailed isotope study was undertaken to determine the relative rates of volatilization of lindane and parathion from water in planchets and from flooded soil environments. The studies showed that lindane disappeared faster than parathion from planchets. More rapid loss of both insecticides occurred from water than from chloroform. The loss of lindane and parathion was related to measured losses of water by evaporation. During the 5-day incubation under flooded soil conditions, lindane disappeared faster from open vials than from sealed vials, whereas in nonflooded soils no volatile loss of the insecticide was evident despite water evaporation. Over 5 day incubation under flooded conditions, a greater volatile loss of lindane was recorded in sandy soil than in alluvial soil. This was apparently due to greater adsorption to the soil colloids which decreased the insecticide concentration in the standing water of the laterite soil. Lindane loss was directly proportional to its initial concentration in water under identical conditions of water evaporation. This study shows that considerable loss of soil applied pesticides can occur by volatilization from the standing water in flooded rice fields, particularly under tropical conditions.

76-2125. Choi, K. L.; Que Hee, S. S.; Sutherland, R. G. (Dep. Chem. Chem. Eng., Univ. Saskatchewan, Saskatoon, Saskatchewan, Canada). **2,4-D levels in the south Saskatchewan River in 1973 as determined by a GLC method.** *J. Environ. Sci. Health* B11(2): 175-183; 1976. (17 references)

This paper presents the levels of 2,4-D derivatives obtained using water samples from the South Saskatchewan River in agricultural districts surrounding Saskatoon both at the height of the spraying season and at harvest time when little 2,4-D should be detected. The method of analysis used direct GLC analysis and alkaline hydrolysis, followed by *n*-butylation of the liberated free acid after acidification of the alkaline solution, and confirmed by subsequent *n*-octylation. GC/MS was used to confirm the presence of 2,4-D. At the height of the spraying season, 2,4-D was detected at an average level of about 2 μg of acid equivalent of 2,4-D/liter of river water. 2,4-D was not detected at the time of harvest or in river mud samples.

76-2126. Murai, T.; Tomizawa, C. (Natl. Inst. Agric. Sci., Kita-Ku, Tokyo, 114 Japan). **Chemical transformation of *S*-benzyl *O*-ethyl phenylphosphonothiolate (Inezin) by ultraviolet light.** *J. Environ. Sci. Health* B11(2): 185-197; 1976. (13 references)

This paper describes the photodecomposition of Inezin exposed to UV light under laboratory conditions. At the initial stage of irridation two main steps of photodecomposition were noted. One was the cleavage of the P-S bond to produce *O*-ethyl phenylphosphinate and dibenzyl disulfide. The dibenzyl disulfide degraded rather rapidly to produce sulfuric and benzoic acids through toluene-α- sulfonic acid by oxidation. The second step was the isomerization to the thionate, *O*-benzyl *O*-ethyl phenylphosphonothionate, which was gradually oxidized to its oxygen analog, *O*-benzyl *O*-ethyl phenylphosphonate. Under UV light, *O*-ethyl phenylphosphinate, *O*-benzyl *O*-ethyl phenylphosphonate, and *O*-benzyl *O*-ethyl phenylphosphonothionate were fairly stable as compared to Inezin. As hydrolysis products of the parent compound, phenylphosphonic acid, *O*-ethyl hydrogen phenylphosphonate, *O*-ethyl hydrogen phenylphosphonothionate, and benzyl alcohol were detected.

76-2127. Cottrell, A. (Author address not given). **Mercury: the controversy flares anew.** *Mod. Paint Coatings* 66(4): 19-21,24; 1976.

On February 17, 1976, EPA Administrator Russell Train suspended registrations for all alkylmercury pesticides, all other mercury products applied to laundry fabrics and rice seed, and those used in marine antifouling paints. Registrations for all other mercury pesticides, deemed not to create an imminent hazard in the "environment," were cancelled. The decision permits the sale and use of phenylmercurial pesticides formulated on or before February 17. EPA contends that the phenylmercurials used in pesticides are converted to methylated mercury in lake, river, and ocean sediments. Suppliers of mercurials contend, however, that only a part of the phenylmercurials present in water are methylated and that methyl mercury is demethylated by bacteria into less harmful compounds. Train's linking of methyl mercury poisonings in Japan, Iraq, and New Mexico to the case against phenylmercurial pesticides has also drawn industry opposition. Since these incidents involved the ingestion of large amounts of chemically synthesized methyl mercury, they are felt to be irrelevant to a discussion of the risks of phenylmercurial pesticides.

76-2128. Winnett, G.; Helrich, K.; Menzer, R. E.; Linscott, D. L.; Bourke, J. B.; White-Stevens, R. H. **Pesticide residues in or on raw agricultural commodities.** *N. J. Agric. Exp. Stn. Bull.* No. 834, 1973, 107p. (132 references)

Pesticide residue levels in and on various vegetable crops, fruit crops, forage, and animal products were determined as part of a Northeast Regional Research Project (NE-36) conducted cooperatively by the State Agricultural Experiment Stations of Maryland, Massachusetts, New Jersey, New York, Rennsylvania, and Rhode Island. Since its inception in 1956, NE-36 had used a conference and committee approach to regional pesticide research. It has studied many pesticides which have been registered for agricultural use as well as many compounds which have not been proven effective or safe. The objectives of the

project are: to determine the amounts of residues and degradation products resulting from the use of pesticides and plant growth regulators applied to soil, plants, and animals; to determine proper management practices for the effective use of pesticide materials and plant growth regulators that will result in minimum residues on the marketed product without undue contamination of other commodities, the soil, water, and other parts of the environment; and to develop and standardize sampling and analytical methods for the determination of residues and degradation products resulting from the agricultural use of these materials. NE-36 disseminates information to state and federal agencies and to other regional research technical committees.

76-2129. Martin, N. A. (Entomol. Div., DSIR, P.B., Auckland, New Zealand). **Effect of four insecticides on the pasture ecosystem. I. Insecticide residues, soil moisture and bulk density, and herbage yield and composition.** *N. Z. J. Agric. Res.* 17(4): 485-494; 1974. (30 references)

The effects are described of insecticide treatment on soil moisture and bulk density, and on herbage yield and composition. The insecticides used were fensulfothion, fenitrothion, carbofuran, and DDT, each applied as commercial granules at the recommended rates for the control of grass grub, 2.24 a.i./ha. Most of the insecticide residues remained in the top 30 mm of the soil. Only a trace of fenitrothion was present in the soil 7 weeks after treatment, whereas after 30 weeks some fensulfothion and most of the DDT residues were still present. These insecticides had no detectable effect on soil moisture and bulk density, or on herbage yield and species composition. The results of this trial have shown that insecticides applied to the surface of a pasture, even under favorable conditions, are unlikely to penetrate much below 30 mm. The study has also shown that on a silty loam soil, in a small plot trial where no major pests were present, fensulfothion, fenitrothion, carbofuran, and DDT did not have any medium-term effects on the soil or on plant growth.

76-2130. Metcalf, R. L.; Lu, P. Y.; Kapor, I. P. (Dep. Entomol., Illinois Univ., Urbana, IL). **Environmental distribution and metabolic fate of key industrial pollutants and pesticides in a model ecosystem.** *Natl. Tech. Inform. Serv.*, PB-225 479, 1973, 80 p. (37 references)

The contamination of water supplies with toxic substances liberated into the environment either accidentally as industrial or household wastes or purposefully as pesticides is an important environmental problem. Organochlorine pesticides have become the prototype microchemical pollutants as they have very low water solubilities, high lipid solubilities, and thus a high partition coefficient in lipid/water. They accumulate readily in food chain organisms, and have a delayed onset of problems in environmental toxicology such as reproductive difficulties in various organisms, especially those with higher positions in food chains. Micropollution by these pesticides has become ubiquitous and will be a problem for years to come. Radiolabeled contaminants used in a lab model ecosystem provide a method for determining the degradative fate of potential pollutants, indicate their toxic effects for food chain organisms, and measure their ecological magnification in food chains. Many insecticides and herbicides have been evaluated as radiotracers in such model ecosystems with a seven element food chain. Such technology provides a simple and informative screening technique for determining potential environmental pollutants and for selecting suitable biodegradable molecules for industrial development.

76-2131. Bennett, G. W. (Dep. Entomol., Purdue Univ., West Lafayette, IN). **Evaluation of diazinon residues in human food.** *Pest Contr.* 44(6): 33-34; 1976. (2 references)

A 1.0% diazinon solution was applied to selected areas of a grocery store, bakery, and cafeteria for cockroach control. Samples of food were tested for residues before and at various times after spraying. Diazinon residues were less than 0.05 ppm in wrapped and unwrapped samples of apples and tomatoes, and residues greater than 0.05 ppm were found in unwrapped, but not wrapped, meat samples. No diazinon residues were found in any of the cafeteria foods, and no residues were found in bakery samples taken 30 min after treatment. Bakery samples taken 12 hr after treatment contained average residues of 0.21 ppm. The results indicate that a residual insecticide can be used around exposed food with minimum insecticide movement into the food. However, label directions should be followed explicitly and good practice guidelines in insecticide application should be followed. Diazinon now has label clearance for use as a 1.0% crack and crevice treatment in food handling establishments.

76-2132. Kearney, P. C.; Plimmer, J. R.; Wheeler, W. B.; Kontson, A. (Agric. Environ. Quality Inst., Agric. Res. Serv., Beltsville, MD 20705). **Persistence and metabolism of dinitroaniline herbicides in soils.** *Pestic. Biochem. Physiol.* 6(3): 229-238; 1976. (13 references)

Air-dried Matapeake loam soil was treated with 10 ppm of trifluralin, profluralin, dinitramine, butralin, fluchloralin, or chlornidine in benzene plus 1 μCi of ^{14}C-labeled herbicide. The soil moisture was maintained at 70% field capacity and the samples were incubated for 3-7 mo. Herbicide mass spectral analysis and residues were determined by high-pressure liquid (HPLC), thin-layer (TLC), and gas-liquid (GLC) chromatography. Dinitramine was rapidly degraded during the first 5 mo, and butralin and chlornidine were less persistent than fluchloralin, profluralin, and trifluralin after 7 mo. The latter three herbicides were similar in persistence and binding properties. The original herbicide was the major ex-

tractable product detectable in the soil in each case and at all sampling times. Dealkylated and cyclic derivatives of the parent herbicides were detected as metabolites. The cyclic products included benzimidazole derivatives of dinitramine, trifluralin, and fluchloralin, a morpholine derivative of chlornidine, and a quinoxaline derivative of fluchloralin. A unique metabolite of butralin was derived from the parent compound by the loss of one nitro substituent. All of the soil extracts contained some very polar material near the origin on the TLC plates and there were minute quantities of nonpolar materials on most plates.

76-2133. Walker, A. (Nat. Vegetable Res. Stn., Wellesbourne, Warwick, England). **Simulation of herbicide persistence in soil. III. Propyzamide in different soil types.** *Pestic. Sci.* 7(1): 59-64; 1976. (8 references)

The effects of soil temperature and soil moisture content on the rate of degradation of propyzamide in five soils were examined under controlled laboratory conditions. Half-lives in soils incubated at field capacity varied from 23 to 42 days at 25°C and from 63 to 112 days at 15°C. The variation in half-life at 25°C and 50% of field capacity was from 56 to 94 days. When the laboratory data were used in conjunction with the relevant meteorological records and soil properties in a computer simulation program, predicted degradation curves for propyzamide in four of the soils in microplots were in close agreement with those observed. Use of the program to predict residues of propyzamide in the fifth soil at crop maturity in a series of field experiments concerned with continuity of lettuce production gave values fairly close to those observed when appropriate corrections were made for initial recoveries. (Author abstract by permission)

76-2134. Ben-Aziz, A.; Shabi, E.; Aharonson, N. (Pestic. Chem. Residue Res. Lab., ARO, Volcani Cent., Bet Dagan, Israel). **Persistence and distribution of three benzimidazole fungicides in pear trees sprayed for the control of *Venturia pirina*.** *Phytoparasitica* 2(2): 117-125; 1974. (12 references)

Benomyl, carbendazim, and methylthiophanate were applied to 6-yr-old Spadona pear trees at rates of 150 ppm a.i./ha (benomyl and carbendazim) and 210 ppm a.i./ha (methylthiophanate) using an airblast sprayer. A total of seven treatments were given between March and May. Chemical analysis and bioassays showed that basal leaves sprayed more than once with benomyl contained 50 ppm methyl benzimidazole carbamate (carbendazim, MBC) 4 days after the last spray. Seven months later, 8 ppm MBC was detected in the basal leaves, just before normal abscission. In the apical leaves, the fungicide levels were lower than in the basal leaves. Analysis of the basal leaves showed MBC residues 5 and 7 months after the last treatment with benomyl, carbendazim, or methylthiophanate were very similar. Bound MBC was not detected in the leaves and only low concentrations of 2-amino benzimidazole (2-AB) were detected. There was no translocation of the fungicides from treated leaves to new young leaves. Three weeks after the last benomyl treatment, the level of MBC in the pear fruit was 0.4 ppm, about 85% of the fungicide being found in the peel and only 15% in the pulp. Fruits heavily infected with pear scab consistently contained a lower concentration of MCB than the less heavily infected fruits.

76-2135. Uhnak, J.; Szokolay, A.; Sackmauerova, M.; Madaric, A. (Res. Inst. Hyg., Bratislava, Czechoslovakia). **Zhodnotenie obsahu rezidui DDT a HCH v mliecnom tuku vzhladom na pouzivanie tychto insekticidov v polnohospodarstve na slovensku. [Evaluation of the content of DDT and HCH residues in milk fat in view of the application of these insecticides in agriculture in Slovakia.]** *Pol'nohospodarstvo* 20(6-7): 527-538; 1974. (18 references) (Slovak)

Results are presented of determinations of DDT and HCH (BHC) in milk fat in four different dairy centers in Slovakia. Gas and thin-layer chromatography were used. Large differences in amounts of pesticide residues were found in the different areas corresponding to the quantities of pesticides used. In each area the amounts found in dairy products have decreased from levels found in 1968-1969. The principal causes of insecticide residues in dairy products are incorrect use, formulation, timing, and wind drift.

76-2136. Marsh, J. R.; Minear, R. A. (Dep. Environ. Eng., Illinois Inst. Technol., Chicago, IL 60616). **Distribution and levels of lead and arsenic in Grand Traverse Bay, Lake Michigan bottom sediments.** *Prepr. Pap. Natl. Meet. Div. Environ. Chem. Am. Chem. Soc.* 13(2): 1-4; 1973. (5 references)

A study was conducted to determine the lead and arsenic concentrations and distributions in the bay sediments of Grand Traverse Bay, Michigan. Up to 114,000 pounds of lead arsenate pesticides have been applied each year to the 17,000 acres of sweet and tart cherry orchards in this area. Sediment samples were taken from the top three inches at 18 sampling sites. Arsenic concentrations ranged from 0.2 to 8.5 mg As/kg sediment, dry weight, with yearly averages ranging from 2.3 to 3.5 mg/kg. In general, the concentrations seemed to correspond to the use patterns, although the type of sediment did exert considerable influence. It is suggested also that there is another source of lead other than the pesticides. The lead concentrations are currently being related to use and runoff patterns in the area near the orchard soils by comparing orchard soil, river bed, and swamp sediments around the bay.

76-2137. Hargrave, B. T.; Phillips, G. A. (Dep. Environ., Fish. Mar. Serv., Marine Ecol. Lab., Bedford Inst. Oceanogr., Dartmouth, Nova Scotia, Canada). **Adsorption of ^{14}C-DDT to particle surfaces.** *Proc. Int. Conf. Transp. Persistent Chem. Aquat. Ecosyst.* 2: 13-18; 1974. (20 references)

Radiochemical uptake experiments were performed to quantify the importance of pesticide concentration and particle size for the uptake of DDT by various types of particles. When particles of various types and sizes were exposed to DDT concentrations approaching saturation levels (1.2 μg/l), all particles rapidly equilibrated with the ambient DDT concentrations in solution at equilibrium and that on particle surfaces for all particle sizes examined. Increases in concentrations beyond saturation levels were not paralleled by additional uptake by the particles and in many cases, when sufficiently high DDT concentrations were achieved, uptake reached an asymptotic level resembling a Langmuir isotherm. Particles of increasingly smaller diameter adsorbed greater amounts of DDT at similar concentrations, and uptake at 1 μg/l was inversely related to particle diameter on a logarithmic basis. Differences in organic content associated with various particle types were not reflected in different rates of adsorption. Adsorption was decreased 20% in the presence of 1% buffered formalin solutions.

76-2138. Huang, J. C. (Dep. Civil Engineering, Univ. Missouri-Rolla, Rolla, MO). **Water-sediment distribution of chlorinated hydrocarbon pesticides in various environmental conditions.** *Proc. Int. Conf. Transp. Persistent Chem. Aquat. Ecosyst.* 2: 23-30; 1974. (4 references)

The water-sediment distributions of DDT, dieldrin, and heptachlor in aquatic systems containing clay minerals and/or humic acids were studied using gas chromatography. Closed, completely mixed, batch system, and open, continuous-flow aquatic model systems were used. All three pesticides were easily and rapidly adsorbed by the clays and humic acid solids; the clays adsorbed the pesticides more rapidly but had a smaller capacity compared with humus solids. The mechanism of adsorption appeared to involve dipole-dipole and ion-dipole interactions as well as hydrogen bonding. The absorptive capacities of the clays varied according to the specific pesticide-clay combination. Pesticide adsorption by both clays and humic acids was relatively irreversible. Temperature, pH, and salt concentration of the water appeared to exert little or no effect on dieldrin sorption and desorption. The rate of pesticidal uptake was also unaffected by the presence of three organic pollutants (glucose, alanine, and stearic acid) and by the presence of filtered sewage.

76-2139. Olsson, M. (Swedish Museum Natural History, Stockholm, Sweden). **Time and space dependence of pollutant levels in aquatic biota.** *Proc. Int. Conf. Transp. Persistent Chem. Aquat. Ecosyst.* 3: 49-60; 1974. (31 references)

Suitable test material is needed to follow the "clean-up" of some accumulating substances in the environment after they have been banned. Pollution and dispersion of pollutants can be analyzed using larger numbers of water samples taken at different times and places and using a standard organism. The mercury levels in biota from the River Morrum were studied after the 1965 Swedish ban on the use of mercury. After the ban, the mercury leve's in the biota decreased with an approximate half-life of 1.5-3.5 yr. Also, the highest mercury levels were found further downstream after the ban than during the pollution process. The dispersion of mercury, DDT, and PCB in pike was also studied in the Archipelago outside Stockholm. The mercury and PCB levels decreased when moving from the inner to the outer parts of the Archipelago, the latter having a good water exchange with the Baltic. Even in the outer regions, however, the mercury levels generally exceeded background levels. Total DDT levels did not vary between the inner and outer Archipelago, although DDE and DDD (TDE) decreased in the same manner as mercury and PCB. The DDE levels were about four times higher than those of DDD in all regions.

76-2140. Oloffs, P. C.; Albright, L. J. (Dep. Biol. Sci., Simon Fraser Univ., Burnaby, British Columbia, Canada). **Transport of some organochlorines in B.C. waters.** *Proc. Int. Conf. Transp. Persistent Chem. Aquat. Ecosyst.* 1: 89-92; 1974. (6 references)

The movement and persistence of DDT, lindane, chlordane, and PCB were studied in natural waters (the Fraser River and Georgia Strait in British Columbia). The test substances were added at 0.1-0.25 ppm to samples of water and bottom sediment and the samples were incubated at 7-9°C for 12 wk. Recoveries from water samples incubated in glass-stoppered flasks were near 100%, and in no instance could any breakdown products or metabolites be detected. Residues were recovered from glass wool plugs when these were used, and during extended incubation further movement from the glass wool into the open atmosphere occurred. The addition of surfactant to the water samples greatly retarded the loss of chlordane during incubation, whereas agitation had practically no effect. In the presence of bottom sediments, the organochlorines moved from the waters into the sediments, not into the water-air interface; some were metabolized there. Thus, bottom sediments can prevent the escape of residues from natural waters into the atmosphere, and movement from water downwards into sediments actually occurs.

76-2141. Brady, H. A. (Res. Soil Sci., Southern Forest Exp. Stn., Forest Service USDA, Pineville, LA 71360). **Picloram and dicamba persistence in forest environments.** *Proc. South. Weed Sci. Soc.* 28: 230-235; 1975. (6 references)

This study evaluated the behavior of picloram and dicamba when sprayed on post oak growing on two different soils in central Louisiana. One of the soils was a typic Paleudult, an excessively drained sandy loam of the Ruston series. The other was a Pilinthaquic Paleudult, a heavy silty clay of the Beauregard series. Twelve trees on each area were sprayed with 250 ml of a water solution containing 4 grams of picloram/tree. One day and again at 2, 4, 6,

and 8 weeks after spraying, samples were taken. Bioassays with cucumber seedlings showed that toxic levels of picloram and dicamba remained in leaves and litter under post oak 8 weeks after spraying with rates of herbicides that produced 70 to 100% topkills of the oaks. Picloram was not toxic under 60 cm in Beauregard or under 30 cm in Ruston soil after a period of 8 weeks. Dicamba toxicity did not occur below 15 cm in the well-drained Ruston or 30 cm in the poorly drained Beauregard soil. Presence of forest litter increased the loss of both herbicides from Ruston but not from Beauregard soils.

76-2142. Uhnak, J.; Sackmauerova, M.; Madaric, A. (Nat. Inst. Hyg., Bratislava, Czechoslovakia). **Vysledky viacroncneho sledovania rezidui chlorovanych insekticidov v mliecnom tuku na Slovensku. [Results of several years of study of chlorinated insecticide residues in butterfat in Slovakia.]** *Prum. Potravin* 27(2): 72-74; 1976. (16 references) (Slovak)

Samples of butterfat were taken monthly from five dairies in Bratislava, Zvolen, Liptovsky Mikulas, Michalovce, and Trebic. Lindane (γ-BHC) was treated separately; α-, β-, and δ-BHC were grouped together. The α- and β-isomer amounts were almost equal. Only the main metabolite of DDT, DDE, was found in more than trace amounts. The ratio of DDT to DDE was 1:1 except in Bratislava where it was 1:2. BHC residues were found in concentrations ranging from 0.270 to 0.640 mg/kg fat. It was found that via milk and dairy products daily human intake of chlorinated insecticides is about 0.07 μg/kg body weight of BHC and about 0.07 μg/kg of DDT. Despite the decreasing use in agriculture of chlorinated insecticides in Czechoslovakia in the last few years, residues in butterfat continue to increase. This seems to be because of their great persistence in the environment, but there is a correlation between the amounts used currently and amounts found in butterfat.

76-2143. Cunat, P. (Inst. Agroquim. Technol. Aliment., Valencia, Spain). **Piretroides insecticidas. [Pyrethroid insecticides.]** *Rev. Agroquim. Tecnol. Aliment.* 13(4): 510-513; 1973. (4 references) (Spanish)

Among the properties of pyrethroid insecticides discussed in this study are the ease with which they are degraded in the environment, as well as their low toxicity to mammals and their high toxicity to fish. Pyrethroids are rapidly degraded by exposure to light and oxygen. Pyrethroids are useful in combating pests which attack agricultural products, because they do not readily contaminate harvests.

76-2144. Greve, P.A.; Wegman, R.C.C. (Rijksinst. Volksgezondheit, Bilthoven, The Netherlands). **Bestimmung und Vorkommen von aromatischen Aminen und ihrer Derivaten in niederlaendischen Oberflaechengewaessern. [Determination and occurrence of aromatic amines and derivatives in Dutch surface waters.]** *Schriftenr. Ver. Wasser Boden Lufthyg. Berlin-Dahlem* 46: 59-80; 1975. (German)

Results are presented of systematic monitorings of water bodies in the Netherlands for aromatic amines by a group-specific colorimetric method and a substance-specific gas-chromatographic method. Both methods revealed the absence or only small traces of pesticides derived from aromatic amines. The Rhine and its tributaries were, however, contaminated with certain aromatic amines in free form (aniline, 2-, 3-, and 4-chloroaniline, 4-bromoaniline, and 3,4-dichloroaniline), which originated most probably from effluents from chemical and other industrial plants. Locally high 3,4-dichloroaniline concentrations, found at one sampling site, were indicative of the use of diuron in that area.

76-2145. Ernst, W. (Inst. Meeresforsch., Bremerhaven, Germany). **Pestizide im Meerwasser—Aspekte der Speicherung, Ausscheidung und Umwandlung in marinen Organismen. [Pesticides in seawater—aspects of storage, elimination, and conversion in marine organisms.]** *Schriftenr. Ver. Wasser Boden Lufthyg. Berlin-Dahlem* 46: 81-92; 1975. (27 references) (German)

Studies on the accumulation, elimination, and metabolism of pesticides in benthic sediment, seawater, and marine organisms are reviewed. The DDT concentrations lie in the lower ppt range in seawater, and in the lower ppb range in benthic sediments, via which the DDT residues are introduced into the trophic chain by such marine organisms as *Polychaeta*. Organochlorine pesticides present in seawater were accumulated by such marine microorganisms as phytoplankton, algae, shellfish, and small crustaceans. Accumulation rates ranging from 4,300 to 37,000 were found for DDT in different marine algae (*Cyclotella nana, Skeletonema costatum, Amphidinium carteri*). The cumulation factor determined for *Dunaliella* sp. in a concentration range of 1-300 ppb in water was 647. Certain species, such as *Pseudopleuronectes americanus*, excrete most of the DDT in intact form.

76-2146. Lahmann, E.; Herzel, F. (Inst. Wasser-, Boden- und Lufthyg. Bundesgesundheitsamtes, Berlin, Germany). **Pflanzenschutzmittel im Regenwasser. [Pesticides in rainwater.]** *Schriftenr. Ver. Wasser Boden Lufthyg. Berlin-Dahlem* 46: 93-100; 1975. (15 references) (German)

Rainwater samples collected by Liesegang-Loebner glass vessels in Berlin-Dahlem between June 1970 and May 1971 were analyzed for organochlorine pesticide residues, and rainwater samples collected at 12 sampling sites in West Germany and West Berlin between April 1973 and March 1974 were analyzed for anilines and urea-derived herbicides. The monthly averages of α-BHC in the rainwater samples in Berlin-Dahlem were 6.1-23 $\mu g/m^2$ or 183-839 ng/l, and the lindane residues ranged from 3 to 13.3 $\mu g/m^2$ or from 46 to 404 ng/l. Certain samples contained DDT (1.9-15.5 ng/l), DDE (6 ng/l), dieldrin (5

ng/l), and aldrin (66 ng/l). Most samples were free from urea-derived herbicides. The herbicide levels were highest in samples taken near chemical plants and in rural areas with intense use of herbicides.

76-2147. Antze, H. H. (Berlin, Germany). **Grundwasserschutzgebiete und Pflanzenschutzmittel. [Groundwater protection areas and pesticides.]** *Schriftenr. Ver. Wasser Boden Lufthyg. Berlin-Dahlem* 46: 101-111; 1975. (21 references) (German)

Recent regulations concerning the protection of groundwater around water wells of waterworks in West Germany are discussed in connection with the use of plant protecting agents. The storage and use of plant protecting agents is explicitly prohibited in zone I (in the immediate surroundings of wells) and in zone II (50-day line), and the use and storage of certain less harmful chemicals may be permitted in zone III (beyond the 50-day flow time). The zones should be designated as a function of the structure and filtration capacity of the ground.

76-2148. Maier-Bode, H. (Rickenbach, Germany). **Versickerungspruefungen nach Merkblatt 37—erste Erfahrungen. [Percolation tests according to directive No. 37.]** *Schriftenr. Ver. Wasser Boden Lufthyg., Berlin-Dahlem* 46: 147-153; 1975. (German)

Preliminary results of percolation tests on 100 pesticides in 150 different formulations from 20 manufacturers according to Directive No. 37 are described. The pesticides include compounds from the groups of organochlorine, organophosphorus, carbamate, dithiocarbamate, phthalate, organotin, halogenated carboxylic acid, aryloxyalkane carboxylic acid, carbamic acid, triazine, and uracil derivatives. Parallel tests with pesticides with percolation rates around 50% showed a spread of 10-20%. The percolating water remained pesticide-free in the case of pesticides which are practically insoluble in water or whose solubility is very low (0.03-400 ppm), regardless of the actual formulation. The formulation had no considerable influence on the percolation rate of the pesticides.

76-2149. Schmidt, G. (Inst. Wasser-, Boden- und Lufthyg. Bundesgesundheitsamtes, Berlin, Germany). **Problematik der Verhaltenspruefung von Pflanzenschutzmitteln im Oberflaechenwasser. [Problems of determining pesticide behavior in surface water.]** *Schriftenr. Ver. Wasser Boden Lufthyg. Berlin-Dahlem* 46: 155-164; 1975. (German)

Experiments are described on the biodegradation and other mechanisms of elimination of pesticides from water. Parathion was studied at 1 ppm in water with 3% activated sludge. With peptone added, 9% of the input quantity was still present after 96 hours, against 48% when no peptone was added. A degradation rate of only 9% was observed in tap water in 96 hours. The residual quantity of malathion was only 13% in tap water, and less than 1% in water with activated sludge after 168 hours. No degradation of 2,4,5-T was observed in 168 hours, and 85% of the input quantity of 2,4-D and 91% of the MCPA added to the water were found after 168 hours. Atrazine, terbutryn, and desmetryn were not degraded in water. The high rate of degradation of carbaryl in water (96% in 168 hours) was in large part due to codistillation, which was almost exclusively responsible for the quantitative elimination of dichlobenil from water in 168 hours. Preliminary results with "artificial ponds" show the possibility of anaerobic degradation of pesticides in water.

76-2150. Jones, D.; Ronald, K.; Lavigne, D. M.; Frank, R.; Holdrinet, M.; Uthe, J. F. (Dep. Biomed. Sci., Univ. Guelph, Guelph, Ontario, Canada). **Organochlorine and mercury residues in the harp seal (*Pagophilus groenlandicus*).** *Sci. Total Environ.* 5(2): 181-195; 1976. (37 references)

Samples of blubber, liver, kidney, and brain from ten male, six female neonatal, and four lactating female harp seals (*Pagophilus groenlandicus*) were analyzed for DDT, dieldrin, PCB, and total mercury. The methyl mercury levels in the blood were also determined. Biocide deposition was not significantly different in 10-day-old male and female pups, and 10-day-old pups did not differ significantly from those aged 14 days or more in the hepatic biocide levels. However, the residue levels of DDT, TDE, DDE, and dieldrin tended to decrease with age. There was no clear relationship between biocide levels in the 6-18-yr-old lactating adults and the levels in their pups. Younger adults (6-7 yr) had higher levels of PCB and total DDT in their blubber than older females (10-18 yr). Wide intraspecific variation was noted in the organochlorine and mercury residue levels, although pups taken in 1973 had lower organochlorine residues than those taken from the same area in 1971. The results indicate that detectable amounts of organochlorine and mercury residues are able to cross the placenta in the harp seal.

76-2151. Leoni, V.; D'Arca, S. U. (Ist. Igiene "G. Sanarelli", Univ. Roma, Rome, Italy). **Experimental data and critical review of the occurrence of hexachlorobenzene in the Italian environment.** *Sci. Total Environ.* 5(3): 253-272; 1976. (59 references)

This report describes HCB contamination in Italy, as determined by experimental data obtained from 1969 to 1974. Samples were taken of surface waters, soils, fauna, cow's milk, uncooked and cooked total diets, and human fatty tissue. In 120 samples of surface waters, HCB was present in 90% at an average of 2.5 ppt. In the soil, concentrations were 40 ppb, and as much as 7.2 ppm was noted in the livers of dead birds. In 63 samples of whole cow's milk, HCB was found in each sample, at an average level of 4.2 ppb. Total diet studies evaluated an intake of about 4.2 μg/man/day, representing 11-12% of the allowable daily intake. In 28 samples of human fatty tissue, HCB

was detected in each sample at an average level of 0.491 ppm in fresh tissue, or 1.021 ppm referred to the fat extracted. It is concluded that the distribution of HCB in the environment in Italy and the levels found are not consistent with its rather limited uses in the agricultural sector. Thus, it is likely that there are other sources of the pollution. One of the possible other sources of this poliutant may be the presence of HCB as an impurity in other pesticides.

76-2152. Ochiai, M.; Ohotomi, M.; Ambe, Y.; Shinohara, H.; Hanya, T. (Tokyo Metropolitan Univ., Fac. Sci., Dep. Chem., Tokyo, Japan). **Secular variation of BHC in the paper of books.** *Sci. Total Environ.* 5(3): 273-276; 1976. (2 references)

The concentration of BHC was determined in the paper of books published in different years from 1927 to 1973 in order to consider the distribution, migration, and accumulation of the organochlorine compounds in cities. BHC was widely used from the middle of 1940 to the end of 1960 in Japan. The concentrations of BHC in the paper of books published in this period were relatively high and concentrations in the books published in the following period decreased sharply. BHC could not be detected in books published in 1973. It is conclude l that these results demonstrate a trend in the usage of BHC. BHC was also detected in books published before the chemical was commercially available. It seemed reasonable to assume that these books had been contaminated by BHC during storage. The pathway through which the BHC entered the paper in the books is not known.

76-2153. Goldberg, M. C. (U.S. Geol. Survey, Denver Fed. Cent., Denver, CO 80225). **Reactions in the aqueous environment of low molecular weight organic molecules.** *Sci. Total Environ.* 5(3): 277-294; 1976. (17 references)

Some of the types of materials found in surface and ground waters are identified and a detailed discussion is given of the solution sorption phenomena between dodecylguanidine acetate and minerals commonly found in bed sediments. From 57 stations monitored from 1967 to 1970 the analyses indicated chlorinated hydrocarbons were found in bottom sediments (5 μg/kg-3.5 μg/kg) and in water (5 ng/kg-0.06 μg/l); herbicides were found in water (5 ng/l-1.5 μg/l); and total organic carbon in water was 0.1-51 mg/l. The sorption rates of dodecylguanidine acetate from aqueous solution onto quartz, kaolinite, and a humic soil were contrasted. The results of the study indicate that different mechanisms of sorption are operative in the organic soil and clay systems. Quartz sorbed dodecylguanidine acetate in a comparatively small amount. The organic soil material exhibited a lesser sorption capacity than did kaolinite within the first 5 hr of reaction. However, organic soil sorbed dodecylguanidine acetate from high ionic strength solutions, whereas the kaolinite did not. In environmental situations where these materials are acting as surfaces for sorption, the clay sorption would predominate, mechanistically, if the ionic strength of the solution is low, about 0.01, and the organic soil sorption would predominate when the ionic strength of the solution was about 3.

76-2154. Thomson, J. M.; Cash, G. A. (Dep. Zool., Univ. Queensland, St. Lucia, Queensland, Australia). **Pesticides in commercial fish foods.** *Search* 7(3): 101-102; 1976. (3 references)

In an effort to find pesticide-free food to feed experimental estuarine fishes, it was found that natural foods from the Brisbane River estuary and adjacent waters are contaminated with relatively small but persistent pesticide residues. Nine brands of commercial aquarium foods were also found to contain small but significant levels of pesticides. The residue levels obtained were: BHC, 4-15 ppb; lindane, 1-33 ppb; dieldrin, 2-8 ppb; endrin, 1-5 ppb; DDE, 6-25 ppb; DDD, 6-43 ppb; and DDT, 11-17 ppb. The total pesticide concentration ranged from 45 to 174 ppb, with an average of 90 ppb. The method of Holden and Marsden was used to extract the pesticides from the food and analyses were conducted in a Varian series 2700 gas chromatograph fitted with a scandium nitride electron capture detector. Similar results were obtained in the Queensland Government Chemical Laboratory in the case of three of the foods submitted to them for analysis.

76-2155. LaFleur, K. S. (Clemson Univ., Clemson, SC). **Carbaryl desorption and movement in soil columns.** *Soil Sci.* 121(4): 212-216; 1976. (6 references)

The desorption and movement of carbaryl in selected South Carolina soils was investigated. Carbaryl desorption by added water is rectilinear for the carbaryl/soil range 1-100 μmol/kg. The desorption partition values for l/l soil/water systems ranged from 0.12 for soil with 0.15% organic matter to 3.7 for soil with 5.16% organic matter. Carbaryl desorption from model soils confirmed the feeble restraint imposed by texture and the overriding restraint imposed by organic matter content. The desorption partition is a nonlinear function of soil/water ratios, increasing with increasing ratios. Carbaryl movement in 1-m soil columns wet with one pore volume of water was greatest in soil containing 47% of applied carbaryl in effluent and least in soil with 0% of applied carbaryl in effluent. The equilibrium distribution of carbaryl within soil columns can be predicted from desorption data. The prediction equation uses desorption partition values adjusted for column section soil-water ratios.

76-2156. Nearpass, D. C. (Agric. Chem. Management Lab., Agric. Environ. Quality Inst., Agric. Res. Serv., Beltsville, MD 20705). **Adsorption of picloram by humic acids and humin.** *Soil Sci.* 121(5): 272-277; 1976. (12 references)

An acidic organic material prepared from a low-moor Michigan peat humus soil, two humic acid prepara-

tions separated from the acidic organic material, the humin, and the residue remaining after removal of the humic acids were equilibrated for 24 hr with various picloram plus salt solutions. More picloram was adsorbed by humin than by the organic peat soil humus from which the humin was prepared. However, less picloram was adsorbed by the humic acids extracted from the organic material. The pH-dependent adsorption for all preparations agreed with the concept that the adsorption of picloram is largely that of uncharged (or dipolar) molecules. The effects of salt concentration on adsorption were largely those due to the exchange of hydrogen into the solution, causing a shift in equilibrium toward the molecular form. Anion exchange did not appear to occur with the organic matter preparations. Salting out occurred with 0.1 *M* salt solutions. A slight decrease in adsorption took place at pH values below 1 and this was explained by the fact that picloram becomes diprotonated in this pH region, forming cations which cannot compete with hydrogen ion for adsorption sites.

76-2157. Morgeli, B. (Author address not given). **Adsorption in water and waste-water treatment.** *Sulzer Tech. Rev.* 58(1): 28-32; 1976. (4 references)

The technology is discussed of water purification by adsorption on activated carbon and other adsorption media. Conventional treatment processes such as filtration, oxidation, and chemical disinfection eliminate certain substances such as dieldrin and heptachlor epoxide from the water only poorly. However, in pilot-scale tests using three different carbon absorbers to remove lindane by adding the absorbers into raw water in concentrations of 50 ppb, after a relatively short contact time of about 3 min, carbons A and C showed a high adsorption power. Medium-grade activated carbon seemed to be less suitable for the purification of lindane-containing waters.

76-2158. McCormick, A.; De Vargas, G. Z.; Rozo, M. (Inst. Invest. Technol., Bogata, Colombia). **Investigacion sobre residuos de plaguicidas en productos agricolas. [Investigation of pesticide residues in agricultural products.]** *Tecnologia* 16(87): 7-19; 1974. (11 references) (Spanish)

The intent of this study was to assess the degree to which organochlorine pesticides are found in several basic food items in the diet of the Colombian population (samples were taken from a municipal market in Bogota). Methods used to detect and determine pesticide residues were those established by the Food and Drug Administration (FDA) of the United States. Quantitative determination of BHC, heptachlor, aldrin, chlordane, dieldrin, endrin, DDT, and toxaphene residues was performed by gas chromatography with a Varian chromatograph and an electronic detector with a tritium source. The presence of pesticides in foodstuffs (in ppm) was evaluated with respect to standards of tolerance set by the FDA and FAO-WHO. Data is compiled in a number of tables and is summarized within the text. Recommendations as to areas in need of further investigation are also presented. Organochlorine pesticides represented 30.2% of the total consumption of insecticides in Colombia, while 69.8 per cent were phosphate and carbamate compounds.

76-2159. Sodergren, A. (Dep. Anim. Ecol., Univ. Lund, Lund, Sweden). **Transport, distribution, and degradation of DDT and PCB in a South Swedish lake ecosystem.** *Vatten* 29(2): 90-108; 1973. (42 references)

Transport, distribution, and degradation of DDT and PCB were studied in Lake Havgard, which is representative of unpolluted lakes in South Sweden. For 2 yr the input to the lake was measured by a fallout technique to be 9 and 18 g of DDT and PCB, respectively. The major fraction of residues was associated with particulate matter in the atmosphere. DDT and PCB were taken up by many organisms. However, their accumulation in the higher trophic levels was of limited magnitude. Concerning degradation patterns, *p,p'*-DDT was the dominating compound but in every organism analyzed, the metabolites *p,p'*-DDE and *p,p'*-DDD were the most significant residues. No conclusive evidence of degradation was found for PCB. The ratio of DDT:PCB calculated for fishes sampled during 1968 to 1971 gradually decreased, perhaps indicating a decreasing input of DDT and/or an increasing input of PCB.

76-2160. Lozhkin, E. F.; Teleugaliev, T. M. (Alma-Ata Institute of Veterinary Sciences, Alma-Ata, USSR). **DDT v myase ryb. [DDT in the meat of fish.]** *Vestn. Skh. Nauki Kaz.* 17(5): 112-113; 1974. (Russian)

Ten samples of carp muscle from a fishery near Alma-Ata were analyzed for residues of DDT and its metabolites, and hexachlorocyclohexane (BHC) by thin-layer chromatography. DDT residues, ranging from 0.2 to 2.5 mg/kg, were found in 9/10 samples, DDD (TDE, 0.1-0.8 mg/kg) in 4 samples, DDE (0.1-0.4 mg/kg) in 7 samples, and hexachlorocyclohexane (0.1 mg/kg) in 3 samples. The residue levels increased with the age of the carp.

76-2161. Zhulenko, V. N.; Smirnova, L. A. (Moscow Technological Institute of Meat and Dairy Industry, Moscow, USSR). **Vliyanie tekhnologicheskikh protsessov na ostatochnye kolichestva pestitsidov v myasoproduktakh. [Effect of technological processes on the pesticide residues in meat products.]** *Veterinariya (Moscow)* 2: 94-96; 1976. (Russian)

The effect of different technological processes on the mercury and organochlorine pesticide residue levels was studied in processed meat products. The meat and by-products used in the experiment were from a calf that had been fed granosan (0.1 mg/kg of ethylmercury chloride daily) for 20 days, and then granosan with DDT (0.6 mg/kg/day) and lindane (0.3 mg/kg) for 20 more days. Processing to such products as liver paste, liverwurst, raw smoked sausage, and kidney in tomato sauce caused no

significant reduction in the mercury residue, while the heat treatment involved in the technological process (boiling in water or blanching in 2% sodium chloride solution for 5 minutes) caused considerable reduction of the DDT and lindane residue levels. Baking, steaming, smoking, heating, and sterilization did not reduce the organochlorine pesticide contents.

76-2162. Jamet, P.; Piedallu, M. A. (Station de Phytopharmacie, CNRA, Versailles, France). **Etude de l'adsorption et da la desorption de la pyrazone (amino-5-chloro- 4-phenyl-2 2H pyridazinone-3) par differents types de sols. [Study of the adsorption and desorption of pyrazon (5-amino- 4-chloro-2- phenylpyridazin-3 (2H)-one) by different types of soil.]** *Weed Res.* 15(2): 113-121; 1975. (11 references) (French)

A study was made of the adsorption and desorption of pyrazone on various types of soil. A labeled molecule (5-amino- 4-chloro-^{14}C-*l*-2- phenylpyridazin-3(2H)-one) was employed, using an agitation method, for the study. The adsorption and desorption isotherms were determined corresponding to Freundlich's equation. Analysis of these demonstrated that adsorption of pyrazon was a reversible phenomenon. However, it was noted that desorption took place more slowly than did adsorption. Through statistical analysis of these results it was shown that a positive and highly significant correlation existed between the quantity of pyrazon adsorbed and the amount of organic matter in the soil.

76-2163. Helweg, A. (Dep. Bacteriol., State Lab. Soil Crop Res., 2800 Lyngby, Denmark). **Degradation of ^{14}C-maleic hydrazide in soil as influenced by adsorption on activated carbon.** *Weed Res.* 15(2): 129-133; 1975. (9 references)

Experiments were conducted to investigate the effect of adsorption on degradation. The evolution of $^{14}CO_2$ from soil containing ^{14}C-labeled maleic hydrazide was used to determine the decomposition. An air-dried sandy loam soil of pH (H_2O) 7.1, 2.9% humus, 6.9% clay, and 13.4% silt was used in the study. The addition of 200, 1000, and 5000 ppm of ^{14}C-maleic hydrazide markedly delayed the decomposition of the herbicide. After 4 months of incubation, almost equal amounts of $^{14}CO_2$ were evolved from all treatments, indicating that most of the adsorbed herbicide had gradually decomposed. The degradation rate of maleic hydrazide followed first-order reaction kinetics, but the larger the concentration of carbon the more the degradation rate approached zero-order kinetics. The degradation rate of maleic hydrazide was not delayed when it was added to soil containing activated carbon which previously had been incubated for 4 months. Therefore, the activated carbon appeared to have lost its capacity to adsorb maleic hydrazide during its incubation. A linear correlation between the logarithm of the amount decomposed and the logarithm of the carbon concentration was demonstrated. An equation for this correlation was suggested.

76-2164. Marriage, P. B.; Saidak, W. J.; Von Stryk, F. G. (Res. Stn., Agric. Canada, Harrow, Ontario, Canada). **Residues of atrazine, simazine, linuron and diuron after repeated annual applications in a peach orchard.** *Weed Res.* 15(6): 373-379; 1975. (19 references)

In this study the persistence of simazine, atrazine, diuron, and linuron was examined in a peach orchard where these herbicides had been applied annually at 4.5 kg/ha from 1963 to 1971 to the same uncultivated plots. Determination of the residues present in 1969—1971 was made using bioassays which were confirmed in the final year by chemical analysis. Significant accumulation of herbicides was not observed in these tests. The maximum residue levels, measured in October over the 3 yr, were 7.3 kg/ha for diuron, 3.8 kg/ha for linuron, 1.6 kg/ha for simazine, and 0.4 kg/ha for atrazine in the top 15 cm of the soil profile. Simazine and atrazine showed rapid decreases in amounts after treatment, but diuron and linuron were degraded more slowly. Measurable residues of all herbicides were confined to the top 15 cm of the soil, with the majority concentrated in the top 5 cm. Oats were planted from 1972 to 1974 to chart the disappearance of the chemicals. All herbicides caused highly significant yield decreases in 1972, atrazine causing the least (38%) and diuron the most (86%) reduction. Diuron reduced oat yield in 1973 and caused a highly significant decrease in the weight of young oat plants in 1974.

76-2165. Que Hee, S. S.; Sutherland, R. G. (Dep. Chem., Univ. Saskatchewan, Saskatoon, Saskatchewan, Canada). **Volatilization of formulated butyl esters of 2,4-D from pyrex and leaves.** *Weed Sci.* 23(2): 119-126; 1975. (11 references)

The volatilities of iso and normal butyl esters of 2,4-D [(2,4-dichlorophenoxy)acetic acid] in a commercial formulation applied as thin films on pyrex and as aqueous droplets on pyrex and leaf surfaces increased directly with the available surface area/applied dose ratio (Q), and inversely with the adsorptive and absorptive characteristics of the surface, under the same conditions of temperature (39 ± 1.5°C), relative humidity (RH) (0%), geometry, airflow rate (750 ± 7 ml/min) and light. Partial pressures and rates of volatilization were computed. Herbicide in droplets below 200μ in diameter tended to volatilize faster than it penetrated, but the reverse occurred above this diameter. (Author abstract by permission)

76-2166. Comes, R. D.; Frank, P. A.; Demint, R. J. (Irrigated Agric. Res. and Ext. Cent., Prosser, WA 99350). **TCA in irrigation water after bank treatments for weed control.** *Weed Sci.* 23(3): 207-210; 1975. (9 references)

Concentrations of TCA (trichloroacetic acid) were determined in irrigation water after the sodium salt formulation had been applied to the banks of dry canals at 82 kg/ha for weed control. When the canals were refilled about 5 months after treatment, maximum concentrations of TCA detected in the water ranged from 53 to 297 ppb.

The concentration was highest during the first hour of waterflow. The maximum concentration increased at a rate of 26 to 30 ppb/km of canal treated, unless surface runoff or reduced flow volumes caused abnormal flows in the canals. With one exception, no TCA was detected in the water after 4 hr of flow. (Author abstract by permission)

76-2167. Miller, J. H.; Keeley, P. E.; Carter, C. H.; Thullen, R. J. (Agric. Res. Serv., Shafter, CA 93263). **Soil persistence of trifluralin, benefin, and nitralin.** *Weed Sci.* 23(3): 211-214; 1975. (25 references)

Trifluralin (α,α,α-trifluoro- 2,6-dinitro- *N*,*N*-dipropyl- *p*-toluidine), nitralin [4-(methylsulfonyl)- 2,6-dinitro-*N*,*N*- dipropylaniline], and benefin (*N*-butyl-*N*-ethyl-α,α,α- trifluoro-2,6- dinitro-*p*-toluidine) were applied at two rates for five consecutive years (1966 through 1970) as soil-incorporated treatments to cotton (*Gossypium hirsutum* L. 'Acala SJ-1') before the preplanting irrigation. Repeated applications did not significantly affect stand or yield of cotton. Soil samples taken at a depth of 120 cm showed that herbicide residues were confined to the tilled zone of soil (upper 30 cm), and about 80% of the residue was in the upper 15 cm of soil. The amount of herbicide residue found annually in the upper 15 cm of soil varied with herbicide and with season. Residues 15 months after final application had been reduced markedly, but were still sufficient to injure sensitive crops. Residues at 30 months had been reduced to innocuous amounts. (Author abstract by permission)

76-2168. Sprankle, P.; Meggitt, W. F.; Penner, D. (Monsanto Co., St. Louis, MO 63166). **Adsorption, mobility, and microbial degradation of glyphosate in the soil.** *Weed Sci.* 23(3): 229-234; 1975. (16 references)

Glyphosate [*N*-(phosphonomethyl) glycine] was readily bound to kaolinite, illite, and bentonite clay and to charcoal and muck but not to ethyl cellulose. Fe(III) and Al(III)-saturated clays and organic matter adsorbed more glyphosate than Na(I) or Ca(II)-saturated clays and organic matter. Glyphosate appears to be bound to the soil through the phosphonic acid moiety as phosphate in the soil competed with ^{14}C-glyphosate for adsorption sites. Glyphosate mobility in the soil was very limited and was affected by pH, phosphate level, and soil type. The ^{14}C-glyphosate was biodegraded in soil to $^{14}CO_2$, possibly by co-metabolism. Potentiometric titrations of the compound gave pKa values of 2, 2.6, 5.6, and 10.6. (Author abstract by permission)

76-2169. Baldwin, F. L.; Santelmann, P. W.; Davidson, J. M. (Arkansas Agric. Ext. Serv., Little Rock, AR 72203). **Prometryn movement across and through the soil.** *Weed Sci.* 23(4): 285-288; 1975. (8 references)

Specially constructed runoff plots were used to study the effect of simulated rainfall intensity, antecedent soil moisture, and subsequent rainfall on prometryn [2,4-bis(isopropylamino)- 6-methylthio-*s*- triazine] movement across and through a field soil with a 1% slope. The first cm (45.4 L) of runoff was collected and subdivided. The initial 3.8 L of runoff water generally contained a higher concentration of prometryn than did a composite from the next 41.7 L. The sediment contained a higher prometryn concentration than did the runoff water. However, due to the greater volume of water lost compared to sediment, over 90% of the prometryn lost was in the water fraction. When prometryn was applied to a dry soil and rainfall simulated, runoff losses of prometryn were 0.5% or less of the total amount initially applied. The first runoff producing simulated rainfall caused the largest prometryn losses, but prometryn could not be detected in the runoff 1 month subsequent to application. Prometryn was never detected at soil depths greater than 5 cm. Prometryn runoff was greater from plots in which the soil was wet at the time of application. (Author abstract by permission)

76-2170. Robinson, E. L. (Field Crops Utilization and Marketing Res. Lab., Agric. Res. Serv., Athens, GA 30604). **Arsenic in soil with five annual applications of MSMA.** *Weed Sci.* 23(5): 341-343; 1975. (11 references)

Arsenic residues in soil were examined annually over a 5-yr period following annual applications of MSMA (monosodium methanearsonate) at 4.4, 9, 13, 18, 22, 36, 72, 144, and 288 kg/ha. Elemental arsenic did not increase in any plot receiving MSMA rates less than 36 kg/ha. Plots receiving 72, 144, and 288 kg/ha of MSMA for 5 yr showed significant increases in arsenic residues. The arsenic residues for the two heaviest treatments (144 and 288 kg/ha) increased significantly during the first 4 yr, but did not increase in the fifth year. (Author abstract by permission)

76-2171. Ketchersid, M. L.; Merkle, M. G. (Dep. Soil Crop Sci., Texas A&M Univ., College Station, TX 77843). **Persistence and movement of perfluidone in soil.** *Weed Sci.* 23(5): 344-348; 1975. (16 references)

Perfluidone (1,1,1-trifluoro- *N*-[2-methyl-4-(phenylsulfonyl) phenyl]-methanesulfonamide) was chemically stable for 2 to 3 weeks on the surface of three Texas soils either air dry or at field capacity and at temperatures of 22 and 46°C. Perfluidone was susceptible to photodecomposition when applied to glass petri dishes and exposed to ultraviolet irradiation. Perfluidone was readily leached through neutral or slightly alkaline soils, with a tendency toward greater downward movement in soils having low clay and organic matter content. However, leaching was less in an acid loamy sand than in either neutral loamy sand or clay soils. (Author abstract by permission)

76-2172. Colbert, F. O.; Volk, V. V.; Appleby, A. P. (Eli Lilly and Co. Res. Stn., Fresno, CA 93706). **Sorption of atrazine, terbutryn, and GS-14254 on natural and lime-**

amended soils. *Weed Sci.* 23(5): 390-394; 1975. (16 references)

Adsorption of atrazine [2-chloro-4-(ethylamino)-6- (isopropylamino)-*s*- triazine], terbutryn [2-(*tert*-butylamino)- 4-(ethylamino)-6- (methylthio)-*s*-triazine], and GS-14254 [2-(*sec*-butylamino)-4-(ethylamino)-6-methoxy- *s*-triazine] decreased on natural and limed soils as the soil pH increased to pH 8. More reduction in adsorption occurred with GS-14254 than with the other two herbicides. Increased toxicity to barley from GS-14254 was proportional to decreased adsorption by soils. Adsorption-desorption studies with Chehalis sandy loam (Cumulic Ultic Haploxeroll) indicated that extent and strength of adsorption were equivalent for GS-14254 (pKa 4.36) and terbutryn (pKa 4.32) at a soil pH of 5.20. Two soils, Boyce silt loam (pH 9.6, Cumulic Haplaquoll) and Crooked silt loam (pH 8.5, Xerolic Durorthid), adsorbed the *s*-triazines readily in contrast to the soils below pH 8. The high adsorption values were substantiated by the GR_{50} values on barley for atrazine and terbutryn, 10.2 and 13.2 kg ai/ha, respectively. (Author abstract by permission)

76-2173. Stamm, E.; Wilms, H.; Krumm, M. (BIG Rostock, 25 Rostock, DDR). **Untersuchungen zur Quecksilberexposition bei der Anwendung quecksilberorganischer Saatgutbeizen. [Study of the mercury exposure during the application of organic mercury-containing seed dressings.]** *Z. Gesamte Hyg. Ihre Grenzgeb.* 22(2): 91; 1976. (German)

Results of mercury concentration measurements in the air of 7 seed dressing plants in Rostock and in urine samples from workers at these plants are presented. The mercury concentrations in the air exceeded the MAC values (0.01 mg/m³ for wet dressing plants and 0.05 mg/m³ for dry dressing plants), with values ranging from 0.014 to 0.045 mg/m³ in about one third of the plants investigated. After four-weeks of exposure to these concentrations, mercury levels ranging from 2.5 to 13 μg/liter were detected in urine samples from workers, compared to a normal level of 10 μg/liter. The relatively low urinary mercury level is most probably due to the high liposolubility of organomercurials. The workers presented no symptoms other than those of dust exposure. The findings indicate that exposure to such concentrations for 2-4 months yearly can be sustained symptom-free over several years.

76-2174. Engst, R.; Macholz, R. M.; Kujawa, M. (Zentralinst. Ernaehrung, Akad. Wissensch., Bergholz-Rehbruecke, DDR). **Nachweis von Metaboliten des Hexachlorcyclohexan im menschlichen Urin. [Hexachlorocyclohexane metabolites in human urine.]** *Z. Gesamte Hyg. Ihre Grenzgeb.* 22(3): 205-208; 1976. (10 references) (German)

Fresh urine samples from workers exposed occupationally to lindane and other hexachlorocyclohexane isomers were analyzed for BHCs and their metabolites by thin-layer chromatography and gas chromatography. α-BHC, β-BHC, lindane, δ-BHC, hexachlorobenzene, γ-pentachlorocyclohexene, δ-pentachlorocyclohexane, pentachlorobenzene, pentachlorophenol, 2,3,4,5-tetrachlorophenol, 2,3,4,6-tetrachlorophenol, 1,2,3,4-tetrachlorobenzene, 2,4,6-trichlorophenol, 2,4,5-trichlorophenol and/or 2,3,6-trichlorophenol, and/or 2,3,5-trichlorophenol were identified in the urine samples. Further peaks on the chromatogram, located between those of δ-pentachlorocyclohexene and pentachlorophenol, were tentatively identified as pentachlorocyclohexane, tetrachlorocyclohexane, tetrachlorocyclohexene, and other pentachlorocyclohexene isomers.

76-2175. Thielemann, H.; Haase, H. H.; Grahneis, H.; Achtzehn, M. K. (Lehrstuhl Allgemeine Kommun. Hyg., DDR). **Hygienische Bedeutung des DDT-Nachweises in importierten Obst- und Gemusekonserven. [The hygienic significance of DDT detection in imported canned fruit and vegetables.]** *Z. Gesamte Hyg. Ihre Grenzgeb.* 22(3): 213-215; 1976. (9 references) (German)

Two hundred eighty-one samples of raw and processed fruits and vegetables (cucumbers, green beans, green peas, yellow plums, peaches, apricots, strawberries, pears, grapes, apples, tomatoes, tomato juice, peppers, red beets, fruit juice, mixed vegetables, and mushrooms), imported to East Germany from Poland, Rumania, Hungary, the USSR, Bulgaria, Yugoslavia, Albania, Czechoslovakia, mainland China, and North Vietnam were analyzed for residues of DDT. The residues of DDT and other organochlorine hydrocarbons were below the tolerance limits in all samples.

76-2176. Maier, D.; Wendlandt, E. (Author address not given). **Untersuchungen zum Gehalt an Organochlorpestiziden im Bodensee-Uberlinger See. [Studies concerning the concentration of organochlorine pesticides in Lake Constance-Ueberlinger Lake.]** *Z. Wasser Abwasser-Forsch.* 9(2): 53-54; 1976. (8 references) (German)

Raw water and purified water from Lake Constance and Ueberlinger Lake were analyzed for pesticide residues by gas chromatography. Raw water contained 3-3-5 ng of lindane per liter, i.e., far below the maximum allowable level for water to be used for drinking-water preparation. After microsieving, high-speed sand filtration, and ozonization, the lindane content dropped to 1.5 ng/l. The reduction was due mainly to the removal of plankton and microflocculation of organic matter in connection with ozonization. The peak of an as yet unidentified compound was found, but no other pesticides were detected.

76-2177. Wybieralski, J. (Inst. Soil Sci. Fertil., Szczecin, Poland). **Dynamika przemieszczania i zanikania DDT i HCH w modelowych profilach glebowych. [The dynamics of translocation and disappearance of DDT and HCH in model soil profiles.]** *Zesz. Nauk. Wyzsz. Szk. Roln. Szczecinie* 8(32): 365-378; 1971. (15 reference) (Polish)

The displacement and disappearance of DDT and HCH (BHC) in layers of a variety of soils with different physical and chemical properties was determined at three month intervals for 1 year after application of these compounds to the top of the model columns. It was found that both compounds persist in soil for a long time; the greatest residues were detected in the humus layers regardless of type of soil. The rate of displacement was indirectly proportional to the amount of "floatable" particles in the soil. HCH was more evenly distributed and penetrated deeper than DDT. At the borderline between the accumulation layer and the lower layer where heavier soil is formed, an increased absorption of both compounds was noted. In heavier soil the rate of disappearance of DDT was greater than that of BHC while in light soil the reverse was true. DDT had a lower rate (which was probably due to its poorer solubility in water and greater adsorption) suggesting the possibility that contamination of ground water with BHC is greater than contamination with DDT.

76-2178. Piasecki, J.; Wybieralski, J.; Wybieralska, A. (Inst. Soil Sci. Fertil., Szczecin, Poland). **Wpływ wilgotnosci na szybkosc zmniejszenia sie zawartosci DDT i HCH w glebach o roznych wlasciwosciach fizykochemicznych. [Effect of moisture on speed of disappearance of DDT and BHC from soils with different physical and chemical characteristics.]** *Zesz. Nauk. Wyzsz. Szk. Roln. Szczecinie* 8(32): 271-282; 1971. (15 references) (Polish)

BHC and DDT (0.6 g) were added to petri dishes containing 60 g samples of soil which differed in amount of organic carbon (1.74% or 18.8%) but were similar in their physicochemical properties or contained no organic carbon and were dissimilar in their physical and chemical characteristics. All samples were incubated at 30°C ± 0.5°C and relative moisture of either 40 or 95%. Determinations of residues were made at 12 day intervals for 72 days. It was found that an increase in moisture from 40 to 95% speeded up the disappearance of both DDT and BHC in all samples. The decrease in amount of BHC was greater (10.2-33.9% depending on sample) than the decrease in DDT (5.1-21% depending on sample) except in the sample containing 17.8% organic carbon where the opposite was detected (20.4% decrease in DDT as compared to 16.4% decrease in BHC). The effect of moisture on the disappearance of both compounds was less pronounced in samples containing soil than in control samples containing no soil. Among the different soil samples a greater loss of DDT and BHC was noted in those from the humus layers than from those with no humus.

76-2179. Teh-fang, V.; Cheng-jui, Q.; Chen-kai, L.; Ke-ming, C. (Chekiang Univ. Agric., China). **[A preliminary study on the cyanox residue in rice plants.]** *Kun Ch'ung Hsueh Pao (Acta Entomol. Sin.)* 18(1): 25-27; 1975. (11 references) (Chinese)

The control of rice insects such as *Chilo suppressalis* Walker has been aided by the use of cyanox (cyanophos). Rice plants sprayed with cyanox were extracted with chloroform and the resulting fractions were passed through florisil or aluminum oxide columns and evaporated on a water bath. Gas-liquid, thin-layer, and paper chromatography were used to analyze the residues. Results indicated a rapid loss of cyanox residues in rice plants under the influence of such environmental factors as light and rain. (From author abstract)

76-2180. Nagasaki Prefect. Off. (Nagasaki, Japan). **[Present state of pesticide residues.]** *Nagasaki-Ken Kankyo Hakusho (Nagasaki White Paper on Environmental Pollution)* 1973, p. 165-176. (Japanese)

A history is presented of the banning of some pesticides, and the present state is discussed of pesticide application and residues (principally organochlorine insecticides) in agricultural crops, cow's milk, mother's milk, and soil in Nagasaki Prefecture. There was no case in which residues exceeded the established standards in 1971 and 1972. The highest concentration of total BHC in brown rice was 0.063 ppm. The average concentration of β-BHC in cow's milk decreased from 0.661 ppm in 1970 to 0.093 ppm in 1971 and to 0.047 ppm in 1972. In non-farming women's milk in 1970, the highest concentration of total BHC was 0.483 ppm; of β-BHC, 0.480 ppm; and of DDT, 0.055 ppm. In 1971, however, these levels decreased to 0.288, 0.286, and 0.054 ppm, respectively. Average concentrations of total BHC in paddy fields, upland soils, and orchards were 0.022, 0.036, and 0.031 ppm, respectively; the same values for dieldrin were 0.008, 0.035, and 0 ppm; and for endrin, 0, 0.218, and 0 ppm. Handling and use followed national standards for safety.

76-2181. Nagasaki Prefect. Off. (Nagasaki, Japan). **[Environmental pollution by pesticides.]** *Nagasaki-Ken Kankyu Hakusho (Nagasaki White Paper on Environmental Pollution)* 1974, p. 156-166. (Japanese)

After describing the ban of pesticides such as parathion, TEPP, organomercurials, DDT, and BHC, the present status of pesticide residues in agricultural crops, in cow's milk, and in soil was discussed. Pesticide residues such as arsenic, lead, BHC, DDT, parathion, dieldrin, aldrin, endrin, EPN, malathion, diazinon, carbaryl, kelthane (dicofol), chlorobenzilate, fenitrothion, and fenthion were not detected in 86 specimens of agricultural crops of 16 kinds in 1973, except for two cases of lead in strawberry and spinach. The average residues of α-, β-, γ-, and δ-BHC in cow's milk were 0.004, 0.013, 0.001, and 0.000 ppm in 1973. These values were less than one tenth of the tentative upper limit. After experiments of deep tilling to 25-30 cm

below the surface it was found that the contents of dieldrin in soil at 0.15, 15-25, 25-35, and 35-45 cm were 0.006, 0.012, 0.008, and 0.000 ppm. No pesticides were detected in spring 1973. The content of dieldrin in potato harvested from this soil was 0.0022 ppm in the autumn of 1972 and undetectable in the spring of 1973.

76-2182. Hiramatsu, R.; Furutani, F. (Yamaguchi Prefect. Agric. Exp. Stn., Yamaguchi, Japan). [**Persistence of spray deposits of pesticides in tree fruits. The influence of the characteristics of tree fruits on adhesion, drop-off, and permeability of fenitrothion.**] *Noyaku Kagaku (J. Pestic. Sci.)* 3(3): 145-151; 1976. (10 references) (Japanese)

Data was gathered on the persistence of MEP (fenitrothion; dimethyl 4-nitro-*m*-tolyl phosphorothionate), its adhesion to fruit peels, residues in fruit peels and pulp, disappearance from peels, and permeability into peel and pulp. A spray formulation was applied (400 ml insecticide/m^3). MEP deposits were washed away with 100 ml acetone and determinations were made by FTD-gas chromatography. Adhesion was affected by the amount of foliage, by form and bearing of fruits, and by the tissue structure of the fruit peels. Residue adhesion/unit area of fruit grew larger as the fruit grew. Disappearance from fruit was greater with hairy and multiponous fruits (*Prunus mume*) than with lightly haired multiponous fruits (apples). MEP permeability was greater in lightly haired fruits than in hairy multiponous fruits. In the case of grapes, MEP disappearance was greater and permeability lower in thin bunches, and disappearance was lower and permeability higher in thick bunches.

76-2183. Toyama, T.; Tamagawa, S. (Mitsui Toatsu Chem., Inc., Tokyo, Japan). [**Persistence of a herbicide, 2,4,6-trichlorophenyl-4-nitro phenyl ether (MO).**] *Noyaku Kagaku (J. Pestic. Sci.)* 3(4): 178-184; 1976. (15 references) (Japanese)

The persistence of the herbicide mo (CNP, 2,4,6-trichlorophenyl- 4-nitrophenyl ether) in paddy fields was cited from various reports and studies. Its half-life is 6-9 days, depending on the test field, with dose rates ranging from 3 to 9 kg/10 are. Residues were undetectable (less than 0.001 ppm) in brown rice harvested from the treated field. The greatest part of residues in soil consisted of the reduction product, 2,4,6-trichlorophenyl 4-aminophenyl ether. The nitro-ether was also reduced non-microbially in soil to form the amino ether, which in turn was bound to humic acid in soil soon after its formation. Further breakdown pathways of the amino ether were acetylation, formylation, hydroxylation of the amino group, and breakage of the ether bond. The bound amino acid seems to be degraded via the same pathways, according to the residues detected. Translocation of the bound amino acid from soil to plants was negligibly small, and it underwent 50-fold bioaccumulation in fish. Thus the bioconcentrative properties were far below those of BHC, DDT, and PCB. Almost no effect was detected on soil microbes.

76-2184. Okazaki, H. (Pathol. Insect Dep., Inst. Agric. Sci., Minist. Agric. For., Tokyo, Japan). [**Determination of pentachloronitrobenzene residues in Chinese cabbage and soil.**] *Noyaku Kagaku (J. Pestic. Sci.)* 3(4): 201-204; 1976. (10 references) (Japanese)

Chinese cabbage seedlings were transplanted 10 cm from the surface in test fields just after the soil was treated with 20% dust of pentachloronitrobenzene (PCNB) at rates of 20, 30, and 40 kg/10 are. The crops were harvested on the 83rd day after transplanting. Residues were determined by extraction with acetone, partitioning into *n*-hexane, and gas chromatogrpahy. The recovery of fortified compounds was more than 90%. Residue determinations for PCNB and its metabolites, pentachlorothioanisole (PCTA) and pentachloroaniline (PCA), follow (background, 20, 30, and 40 kg/10a PCNB-treated fields): 0.0004, 0.00002, 0.00003, and 0.0002 ppm (PCNB); 0.0006, 0.09, 0.007, and 0.007 ppm (PCTA); and < 0.0002, < 0.0002, < 0.0002, and < 0.0002 ppm (PCA). In soils treated with 40 kg PCNB/10 are, the contents of PCNB, PCTA, and PCA are: 0.03, N.D., and < 0.005 ppm just after application; 0.13, N.D.,and 0.008 ppm 1 mo after; 0.03, 0.004 and 0.0005 ppm 2 mo after; 0.14, 0.0005, and 0.006 ppm 3 mo after; 0.04, 0.007, and 0.007 ppm 6 mo after; 0.11, 0.03, and 0.03 ppm 9 mo after; and 0.03, 0.01, and 0.03 ppm 12 mo after application.

76-2185. Igarashi, H.; Uchiyama, M.; Sato, R. (Cent. Res. Inst., Nissan Chem. Ind. Co. Ltd., Funabashi, Chiba, Japan). [**Absorption, translocation, and metabolism of pentachloronitrobenzene in plants.**] *Noyaku Kagaku (J. Pestic. Sci.)* 3(4): 205-209; 1976. (7 references) (Japanese)

The absorption, translocation, and metabolism of ^{14}C-labeled pentachloronitrobenzene (2-96 μCi/mg) were studied in young corn plants (aerial part 5 cm high) grown in water containing 2 ppm of PCNB. Radioactive material remained in the root portion, with slight translocation to the stem. PCNB was detected in the root along with its metabolites pentachlorothioanisole (PCTA) and pentachloroaniline (PCA), and two unknown compounds, UK_1 and UK_2. The unknowns were found in the acetonitrile-extractable fraction. The formation rate of UK_1 was the greatest. Almost all of the radioactive material which translocated into the stem, even in small amounts, was UK_1. Residue analysis by gas chromatography was unable to detect UK_1. The chemical structure has not yet been elucidated; however, it is hypothesized to be a sulfur conjugate, not a sugar or peptide conjugate with the pentachlorobenzene moiety.

76-2186. Kobayashi, A.; Tsukano, Y. (Pathol. Insect Dep., Inst. Agric. Sci., Minist. Agric. For., Tokyo, Japan). [**Distribution of pesticide residues in solid head leaf vegetables.**] *Noyaku Kagaku (J. Pestic. Sci.)* 3(4): 210-211; 1976. (Japanese)

The hypothesis that the fluctuation of values determined for pesticide residues in solid head leafy vegeta-

bles is due to the number of damaged outer leaves removed prior to the determination was tested. Heads of Chinese cabbage and cabbage were treated with diluted (600x) captan 50 and 80 WP. Captan distribution percentages per leaf group follow (1st, 2nd-3rd, 4th-5th, 6th-7th, and 8th-center leaves): just after application (95.1, 2.6, 1.1, 9.2, and 1.0%); 7 days after (76.4, 22.2, 0.8, 0.2, 0.2, and 0.2%); and 21 days after (82.1, 7.5, 3.3, 2.5, 2.5, 0, and 2.1%). Captan concentrations following application for the above sections were: just after (17, 0.37, 0.18, 0.062, and 0.2 ppm); 7 days after (9.1, 2.7, 9.082, 0.022, 0.020, and 0.020 ppm); and 21 days after (0.22, 0.082, 0.008, 0.007, 0.006, < 0.006, and 0.006 ppm). The test results verified the proposed hypothesis, and some suggestions were given for uniformly determining pesticide residues in solid head vegetables, based on the results.

76-2187. Tsuge, S.; Kazano, H.; Suzuki, K.; Kashiwa, T.; Tomizawa, C. (Agric. Chem. Inspect. Stn., Minist. Agric. For., Kodaira, Tokyo, Japan). [**Biological condensation of α-naphthaleneacetic acid in a model ecosystem.**] *Noyaku Kensasho Hokoku (Bull. Agric. Chem. Inspect. Stn.)* 15: 36-40; 1975. (7 references) (Japanese)

The fate of carboxyl-^{14}C-labeled naphthaleneacetic acid was traced for 33 days in a model ecosystem. The components of the ecosystem were *Ipomoea edulis* leaves, *Spodoptera littora* larvae, rice plant seedlings, *Ceratophyllum demersum* red snails, mosquito larvae, water fleas, and guppies. It was found that, as measured by radioactivity, naphthaleneacetic acid was degraded fairly rapidly. After 33 days, the organisms introduced contained considerable amounts of naphthaleneacetic acid, and the ecological magnification factors in fish and snail were 492 and 169, respectively.

76-2188. Igarashi, M.; Kawahara, T.; Nakamura, H. (Agric. Chem. Inspect. Stn., Minist. Agric. For., Kodaira, Tokyo, Japan). [**Degradation of *N*-methylcarbamate insecticides in soil. Part 1. Relation between degradation of *o*-*sec*-butylphenyl *N*-methylcarbamate and soil properties.**] *Noyaku Kensasho Hokoku (Bull. Agric. Chem. Inspect. Stn.)* 15: 41-47; 1975. (11 references) (Japanese)

The degradation of *o*-*sec*-butylphenyl *N*-methylcarbamate (BPMC) was monitored in four kinds of soil, sandy clayey loam in a paddy field, clayey loam in a paddy field, and yellowish-brown forest soil. It was found that the *N*-methylcarbamate degraded more rapidly under upland conditions than under flooded conditions; the half-life was 20-30 days in the former and 40-60 days in the latter. The time pattern of *N*-methylcarbamate degradation was followed by two methods: cleanup by column chromatography after ethanol extraction and cleanup by TLC after extraction with acetone. There was no difference in the results. No correlation was found between the degradation of *N*-methylcarbamates and the physicochemical properties of the soil such as total carbon content, clay content, CEC, available phosphate, free iron, pH, and absorption coefficient of phosphoric acid.

76-2189. Igarashi, M.; Kawahara, T.; Nakamura, H. (Agr. Chem. Inspect. Stn., Min. Agric. For., Kodaira, Tokyo, Japan). [**Degradation of *N*-methylcarbamate insecticides in soil. Part 2. Relationship between degradation rate and substituents on the benzene ring.**] *Noyaku Kensasho Hokoku (Bull. Agric. Chem. Inspect. Stn.)* 15: 48-52; 1975. (8 references) (Japanese)

The degradation of six *N*-methylcarbamate insecticides, the 1-naphthyl, 2-isopropoxyphenyl, 2-*sec*-butylphenyl, 2-cumenyl, 3-tolyl, and 3,5-xylyl esters, in paddy field soil (sandy-clayey loam) at 25% of maximum moisture capacity was studied. The rate of degradation was greatest for 3-tolyl-, followed by 1-naphthyl-, then by 3,5-xylyl-, 2-cumenyl-, 2-sec-butylphenyl-, and 2-isopropoxyl-phenyl- *N*-methylcarbamate (MTMC, carbaryl, XMC, MIPC, BPMC, and propoxur) in that order. When the rate was represented in terms of substituents on the benzene ring, it was 3-methyl-, benzo-, and 3,5-dimethyl-, showing a relationship with Hammett's constant. It was found that the degradation rate constant of *N*-methylcarbamate insecticides in soil and the hydrolysis rate constant of these compounds in alkaline solution showed a linear relationship for 10 days after their addition.

76-2190. Watanabe, C.; Nishijima, O.; Nakamura, H. (Agric. Chem. Inspect. Stn., Min. Agric. For., Kodaira, Tokyo, Japan). [**Determination of metals residues in crops. Part 2. Residues of manganese in tomatoes due to application of maneb.**] *Noyaku Kensasho Hokoku (Bull. Agric. Chem. Inspect. Stn.)* 15: 53-58; 1975. (3 references) (Japanese)

After spraying a 400 fold diluted solution of 75% maneb WP onto tomato plants at 100 l/10 are, the variation in manganese content of tomato fruits was examined in relation to times of spraying (one, three or ten) and days after spraying (one, three, seven, or fourteen). Manganese was determined by the atomic absorption method and by polarography. It was found that the residual amount increased as the number of applications increased, and that a tendency of the residues to decrease was observed only after 7 days in the case of 10 applications. The standard deviation of the values by the two methods increased as the amount of manganese increased; however, the coefficient of variation did not change. The maximum amount of residue after 10 applications was 4.37, 3.27, 2.19, and 2.20 ppm, respectively at one, three, seven, and fourteen days after the last application.

76-2191. Watanabe, C.; Nakamura, H. (Agric. Chem. Inspect. Stn., Minist. Agric. For., Kodaira, Tokyo, Japan). [**Residue analytical determination of metals in crops. Part 3. Residual amount of lead in commercialized vegetables and fruits.**] *Noyaku Kensasho Hokoku (Bull. Agric. Chem. Inspect. Stn.)* 15: 59-69; 1975. (8 references) (Japanese)

Residue analyses for lead were carried out on commercially grown vegetables and fruits such as turnip, cab-

bage, cucumber, burdock, Japanese radish, onion, seasoning greens, tomato, eggplant, strawberry, persimmon, cherry, pear, grape, and apple. The produce was harvested mainly in the Kanto area and some in Aomori and Fukuoka. Two methods, both supplemented with polarography, were used: one up to March 1969 and the other up to April 1969. After checking, no noticeable difference was recognized in the analytical values obtained by the two methods. The content of lead ranged from 1.13 ppm in apple harvested in Aomori to zero in pear harvested in Chiba. Generally speaking, the residual amount of lead showed species specificity as well as local specificity. The content of lead does not necessary reflect the application of lead containing pesticides, as in apples, but shows absorption from the soil.

76-2192. Fujimoto, Y.; Igarashi, M.; Nakamura, H. (Agric. Chem. Inspect. Stn., Minist. Agric. For., Kodaira, Tokyo, Japan). [**Residue of tetrachloroisophthalonitrile in greenhouse-cultured melons after spraying, fumigation,and evaporation.**] *Noyaku Kensasho Hokoku (Bull. Agric. Chem. Inspect. Stn.)* (15): 70-73; 1975. (1 reference) (Japanese)

Residue analyses were carried out in peel and pulp of greenhouse-cultured melon fruits heated with 2,4,5,6-tetrachloroisophthalonitrile by spraying, fumigation, and evaporation. The dose rates were 3 and 5 spray treatments at 150 l/10 with a 500 fold dilution of the 75% WP, 3 treatments with 16.3 g/81.5 m^3 of 50% fumigant, and 3 treatments with 32.6 g/81.5 m^3 of 28% vapor fumigant. Determinations were carried out one, 2, 4,and 7 days after the last application. The maximal amount of residue in edible pulp was 0.075, 0.015,and 0.006 ppm respectively after spraying, fumigation, and evaporation, while that in the peel was, in the same order, 17.2, 1.09, and 1.0 ppm. The reduction of residue with time in this experiment was not great, and a tendency for translocation from the peel of the pulp could be considered.

76-2193. Yamashita, S.; Nishijima, O.; Kawahara, T.; Nakamura, H. (Agric. Chem. Inspect. Stn., Minist. Agric. For., Kodaira, Tokyo, Japan). [**Residue of pesticides on/in strawberry. Part 2. Residues of quinomethionate on and in strawberries on open field culture.**] *Noyaku Kensasho Hokoku (Bull. Agric. Chem. Inspect. Stn.)* 15: 74-76; 1975. (1 reference) (Japanese)

Strawberries of the Donner variety were sprayed in an open field with a 2500 fold dilution of quinomethionate (oxythioquinox) 25% WP 6 times at a rate of 150 l/10 are once a week and the pesticide residues were determined by gas chromatography one, 3, 7, and 14 days after the last application. The residues were 0.5, 0.35, 0.2,and 0.05 ppm respectively one, three, seven, and fourteen days after application, showing a gradual reduction similar to the pattern of reduction in greenhouse-grown strawberries. The detection limit with the present method of analysis was 0.01 ppm, with recovery of 88.4% when samples were fortified with 0.1 ppm of the pesticide. The results showed that strawberries could be sprayed safely up to 3 days before harvest.

76-2194. Hyg. Lab., Okayama Prefectural Govt. (Okayama, Japan). [**Organochlorine pesticide residues in agricultural crops, dairy products, meat, eggs, and human milk.**] *Okayama-Ken Eisei Kenkyusho Nenpo (Annu. Rep. Hyg. Lab. Okayama Prefect.)* 22: 134-137; 1975. (Japanese)

A tabulation is presented of residue information collected from 1970-1974. Included are: total DDT, total BHC, and polychlorocyclodienes in 33 kinds of agricultural crops, in eight kinds of ocean and freshwater fish, in four kinds of dairy products including cow's milk, in meat (beef, pork, and chicken), and in eggs and human milk. Materials were tabulated without any comment.

76-2195. Saga Prefectural Office (Saga, Japan). [**Status of environmental pollution by pesticides and its countermeasures.**] *Saga-Ken Kankyo Hakusho (Environ. White Paper) Saga Pref.*, p. 359-369. (Japanese)

Pesticide residues in mothers' milk in Saga Prefecture were: α-BHC (average 0.11 ppm, max. 0.24 ppm (in a non-farm woman and min. 0.03 ppm in a farm woman) and total DDT (average 0.03 ppm, max. 0.059 ppm in a non-farm woman and min. 0.0081 ppm), both being lower than in 1974. Pesticide residues in commercial vegetables, in special crops of the Prefecture, and in cow's milk were less than the national standard. However, their existence was detected (lead, dieldrin, aldrin,and arsenic). Residues in cow's milk were decreased year after year; the average content of the specimens collected in March 1975 was 0.0041 ppm β-BHC, 0.0013 ppm total DDT, and 0.0005 ppm dieldrin. Countermeasures for reducing those residues were described.

see also **76-2107**

76-2196. Chow, I.; Bellin, J. (Med. Computer Sci., Downstate Med. Cent., Brooklyn, NY). **Long-term exposure of factory workers to dichlorvos (DDVP) insecticide.** *Arch. Environ. Health* 30(2): 111-112; 1975.

In a letter to the editor the writers question the statistical methodology and conclusions of an article published earlier (Pesticides Abstracts No. 74-1092) concerning the effects of long-term exposure of factory workers to DDVP (dichlorvos). In particular they question the statement that an observed 35% decrease in erythrocyte AChE was not significant because significant trends could be demonstrated for the majority of these values, thus necessitating the rejection of the null hypothesis for random samples (median test, $P \leq .05$). Also questioned was the use of both the Wilcoxon and the median test which assume independent samples when, in the study cited, each worker served as his own control. It is suggested that the observed large decreases in blood cholinesterases on DDVP exposure may well be injurious to the health of the workers, even in the absence of overt clinical symptoms.

76-2197. Carrington, C. B. (Author address not given). **18th Aspen conference—introduction to summary.** *Chest* 69(2): 321-328; 1976. (4 references)

In the course of the conference a paper was presented which described a model of paraquat lesions. Of the two patterns of fibrosis observed in such cases, one is uncommon. The alveolar architecture is quite extraordinarily preserved and so is the capillary bed in the alveolar walls, but the air spaces are filled by a delicate myxomatous connective tissue with innumerable fine collagen fibrils. In such regions there appears to be severe damage to the alveolar epithelial lining, but no morphologically apparent lesions in the capillary endothelium. It is suggested that the epithelial necrosis and flooding of the air spaces by edema fluid somehow resulted in this unusual pattern of fibrosis. Massive epithelial necrosis was not associated with fibrosis in several lung damage models examined.

76-2198. Hearn, C. E. D. (Gillette Ind. Ltd., Isleworth, Middlesex, England). **Pesticides in agriculture.** *Clin. Med.* 83(3): 12-16; 1976. (8 references)

Organophosphorus insecticides irreversibly inhibit acetylcholinesterase by phosphorylation. The level of tissue enzyme activity at which toxic symptoms begin varies with the rate of inactivation and the degree of adaptation to the depressed acetylcholinesterase level. The early symptoms of poisoning are mild and nonspecific, and specific treatment requires atropine administration and, sometimes, artificial ventilation; a cholinesterase reactivator may also be given. Carbamates cause a reversible inhibition of cholinesterase, but symptoms of intoxication disappear comparatively quickly. The chlorinated hydrocarbons are safe in ordinary use, but are persistent, and acute overexposure may cause CNS stimulation. The organomercurial fungicides, which are used as seed dressings, accumulate in the nervous system and cause progressive degenerative changes. The aryl substituted compounds are much less toxic. The herbicide paraquat is safe under even extreme working conditions, and fatalities are generally due to accidental ingestion; diquat is less toxic. No occupational hazard is involved in the agricultural use of growth regulant herbicides. The dinitro herbicides block the oxidative phosphorylation cycles of carbohydrate metabolism, leading to a vast increase in cellular metabolic rate; pentachlorophenol works in the same manner. Treatment consists of immediate cooling of the patient and oxygen administration.

76-2199. Atkinson, B. (Dep. Med., Univ. Mississippi Med. Cent., Jackson, MS). **Organophosphate insecticide poisoning.** *J. Miss. State Med. Assoc.* 17(4): 91-94; 1976. (5 references)

Twelve cases of organophosphate insecticide poisoning are presented. In the first case, a clinical diagnosis of organophosphorus poisoning was suggested by muscular weakness, miosis, hypotension, and immediate response to specific therapy. Patient 2 was treated with gastric lavage and atropine after he was seen ingesting the insecticide. Five other cases involved children playing in sprayed cotton fields: four responded well to atropine and pralidoxime (PAM), but the fifth died with severe pulmonary edema and hemorrhage with extensive bronchopneumonia. Treatment with PAM and atropine also led to the recovery of a patient accidentally doused with parathion in the face and chest, and another patient who ingested diazinon in a suicide attempt. Eleven of the 12 patients thus either worked with organophosphates or lived near fields where it was being used. Poisoning may be suggested by a history of exposure or by the characteristic signs and symptoms. Treatment with atropine and PAM, decontamination, and general support will usually effect a prompt recovery.

76-2200. Fryer, G. (Author address not given). **Unusual egg spoilage in the common frog.** *Naturalist* 926: 105-106; 1973.

The finding of unusually large numbers of spoiled spawn from the common frog (*Rana temporaria* L.) prompted an investigation into the cause of this situation. Previous studies have indicated that pesticides can cause abnormalities and death of tadpoles and that DDT can penetrate newly deposited spawn, but not spawn containing developing larvae, thus giving rise to subsequent temporary hyperactivity and subnormal weights. It is not suggested that the spawn found in this study were exposed to such contaminants in a direct manner, as the remoteness of the sites at which these spawn were found would make this improbable. However, toxic substances have been found in the eggs of raptorial birds such as peregrines and eagles which breed in remote situations. Thus such a cause as pesticide exposure must be kept in mind for the current situation.

76-2201. Okonek, S. (Med. Klin. Univ., Mainz, Germany). **Detection of paraoxon in the blood of patients with severe suicidal nitrostigmine intoxication.** *Naunyn-Schmiedeberg's Arch. Pharmacol.* 293(Suppl.): R68; 1976.

Paraoxon, a metabolite of nitrostigmine (parathion), is the true toxic agent in nitrostigmine intoxications. Hitherto, paraoxon could only be determined in vitro, in animal experiments, or post mortem. Toxicological investigations were performed in 19 patients with nitrostigmine intoxication. Paraoxon was detected in the blood of 5 severely poisoned patients, with the highest paraoxon concentration being 6.3 μg/ml of plasma. In 4 patients paraoxon could be detected only in the acute intoxication phase, and in one case up to 11 days following ingestion. During this phase the patients were comatose, paralytic, and without spontaneous respiration. There were atypical EEG patterns. Catecholamine levels were elevated. No activity of nonspecific cholinesterases or acetylcholinesterase could be measured, and free acetylcholine was present in the blood. Three patients survived. One patient was also treated by hemoperfusion over coated activated charcoal. Both nitrostigmine and paraoxon were readily eliminated from the blood with the aid of this procedure; clearance values were 72.88 ml/min for nitrostigmine and 76.18 ml/min for paraoxon. Paraoxon was detected by gas chromatography with an FID and selective sensitivity for phosphorus-containing substances. Results were confirmed by mass spectrometry using the multiple ion detection procedure. (Author abstract by permission)

76-2202. Van Heijst, A. N. P.; Douze, J. M. C.; Pikaar, S. A. (Nationaal Vergiftigingen Informatie Centrum, Rijks Instituut voor de Volksgezondheid, Bilthoven, The Netherlands). **Nationaal Vergiftingingen Informatie Centrum. [Nationaal Poison Information Center.]** *Ned. Tijdschr. Geneesk.* 120(5): 206-209; 1976. (Dutch)

Data is presented on the activity of the National Poison Information Center in the Netherlands during the period of 1965-1974. Information was requested concerning poisoning with insecticides and pesticides on 3,051 occasions. Ninety-nine adults and 48 children were hospitalized for poisoning with pesticides and insecticides. Documentation, research, and clinical practice are combined in the Poison Information Center. Dithiocarbamate, being hazardous, has been replaced in the therapy of thallium poisonings by Prussian blue. Further studies are being conducted on the therapy of paraquat poisoning.

76-2203. Fournier, E.; Dally, S.; Cambier, J. (Fernand-Widal Hosp., Paris, France). **Neuropathie peripherique probablement due aux insecticides anticholinesterasiques. [Peripheral neuropathies probably induced by anticholinesterase insecticides.]** *Nouv. Presse Med.* 5(11): 718; 1976. (6 references) (French)

A 32-year old female worker, who customarily handled dichlorvos, benomyl, and aldicarb in a closed atmosphere and without respecting safety measures, after one year developed paresis of the arms and legs accompanied by an intense physical asthenia; EMG showed peripheral neurogenic symptoms. After three months away from the insecticides, her cholinesterase rates returned to normal. Although the chemical formulas of organophosphate pesticides are closely related to that of triorthocresylphosphate (TOCP), they have rarely been described as causing peripheral neuropathies except for mipafox, which has been banned. However, electromyographic alterations have been described in exposed workers. Also, the action of these pesticides on motor function could explain the periodic occurrence of intense muscular fatigue in those who handle the chemicals.

76-2204. Montgomery, R. R.; Reinhardt, C. F. (Haskell Lab. Toxicol. Ind. Med., E.I. du Pont de Nemours and Co., Wilmington, DE). **A capsule dose of toxicology.** *Occup. Health Nurs.* 24(5): 7-16; 1976. (21 references)

This review on toxicology defines the terms LD50, LC50, ED50, and EC50 as well as the OSHA and TLV limits on occupational exposure to specific materials. The review also considers the following topics: respiratory pollutants, such as asphyxiants, dusts, and granuloma-producing agents; systemic pollutants including heavy metals; respiratory allergens; carcinogens, including skin and subcutaneous tissue carcinogens, respiratory tract carcinogens, and urinary tract carcinogens; mutagens; embryotoxins and teratogens; fungi and bacteria; skin diseases from contact irritants, sensitizing agents, or biologic agents; and specific eye injuries. Pesticides are included in many of these categories.

76-2205. Takats, G. (Orszagos Birosagi Vegyeszeti Intezet, Budapest, Hungary). **Harm ev foszforsavesztermergezeseinek ertakelese. [Evaluation of organophosphate poisonings in a 3-year-period.]** *Orv. Hetil.* 115(38): 2240-2242; 1974. (Hungarian)

Statistical analysis is presented of lethal poisonings with organophosphorus pesticides between 1971 and 1973 in Hungary. The death toll was 69 in 1971, 53 in 1972, and 93 in 1973. Most victims were men, while children accounted for about 3%. Most pesticide poisonings, either accidental or suicidal, occur during the July-November period, most probably due to careless storage of the pesticide preparations used during the summer season. Both the absolute value and the percentage of poisonings with organophosphorus compounds show an increase, while the incidence of poisonings with preparations that are authorized for large-scale use only is decreasing. Seven cases were due to demeton-*O*-methyl, 8 to phosphamidon, 14 to methyl parathion, 31 to mevinphos, 11 to dichlorvos, 27 to dimethoate, 31 to malathion, and 32 to trichlorfon.

76-2206. Kashyap, S. K.; Gupta, S. K. (Natl. Inst. Occup. Health, Ahmedabad-380 016, India). **Significance of blood cholinesterase enzymes as an index to evaluate exposure to organophosphorus insecticides.** *Pesticides* 10(1): 36-38; 1976. (10 references)

A large-scale investigation was undertaken in India on blood cholinesterase activity to assess the significance of this parameter in safeguarding the health of people who are occupationally exposed to organophosphorus insecticides. Blood cholinesterase levels were measured in 350 normal subjects and in 166 subjects occupationally exposed to organophosphorus insecticides. The results of the study showed that the measurement of blood ChE activity can be an effective index in the control of occupational exposure to these chemicals. A fall of more than 20% in blood ChE should be regarded as significant. In an individual for whom pre-exposure values are not available, it is assumed that red cell activity 20% or plasma activity 33% below the population average offers good indication that that individual has been exposed to cholinesterase inhibitors. There is no general agreement on the degree of ChE inhibition likely associated with toxic manifestations. However, it is safely presumed that toxic manifestations will occur only if ChE activity has fallen well below 50% of its normal value. It is concluded that the usefulness of blood ChE tests as an index in the evaluation of toxicity hazards from organophosphorus insecticides is indisputable.

76-2207. Kontek, M.; Pietraszek, Z.; Marcinkowska, B. (Cardiol. Clin., Inst. Int. Med., Poznan, Poland). **Ocena wplywu przewleklego kontaktu z pestycydami na zapis EEG u pracownikow rolnych. [Evaluation of the effect of long-term contact with pesticides on EEG in farm workers.]** *Pol. Tyg. Lek.* 30(31-32): 1319-1321; 1975. (12 references)

The effects of chronic exposure to pesticides were evaluated in the EEG curves of farm workers. Two to 5 tracings were obtained from the 16 workers examined. The first examination was made some 1 to 2 months after the exposure in 9 cases. In 6 of these 9 cases the tracing at the time was normal, in 2 it showed slight changes, and in only 1 case was this tracing pathological. After 2.5 yr of work with the pesticides, the tracings were normal in 4 cases with slight changes in 2. In 11 workers the EEG tracings were recorded before they started working with the chemicals. In 6 of these 11 cases the tracings were normal and in 5 cases they showed slight abnormalities. EEG tracings were made in 9 workers after they had not been exposed to pesticides for a period of 6 months. In one case a pathological tracing became slightly abnormal and in one case the tracing changed from one with slight abnormalities to a pathological one.

76-2208. Klose, R.; Gutensohn, G. (Inst. Anaesthesiol. Reanim., Fac. Clin. Med. Mannheim, Univ. Heidelberg, Mannheim, Germany). **Behandlung einer Alkylphosphatintoxikation mit gereinigter Serumcholinesterase. [Treatment of alkylphosphate poisoning with purified serum cholinesterase.]** *Prakt. Anaesth.* 11(1): 1-7; 1976. (21 references) (German)

In an attempt to commit suicide, a 26-yr-old man injected himself i.m. with 3.75 gm of demeton methyl. Severe symptoms of poisoning developed after 36 hr, with loss of consciousness and respiratory arrest. Symptomatic treatment failed to improve his condition and he was given injections of purified serum cholinesterase. This was followed by a rise in the patient's cholinesterase levels; the increased activity was accompanied by a rapid and definite improvement in his clinical status. The results demonstrate the usefulness of this method of treatment. Transfusions of fresh blood to counteract the cholinesterase deficiency are inadvisable because of the risk of complications. Substitution therapy with serum cholinesterase must be accompanied by the administration of large doses of atropine.

76-2209. Clayton, R. (Dermatol. Dep., St. Mary's Hosp., London W2 1NY, England). **From fentichlor sensitivity to actinic reticuloid?** *Proc. R. Soc. Med.* 69(5): 379-380; 1976. (4 references)

A male industrial chemist, 48 yr of age, who had been involved in the manufacture of fentichlor for 15 yr, experienced an occasional eczematous rash of the hands. He also had an eczematous rash on the scalp and face two years and one year earlier after exposure to fentichlor and noted swelling and a rash of the face with gross edema of the eyelids requiring treatment with prednisolone. Even though he has avoided contact with the pesticide he continues to have eczematous rashes of the face, neck, and hands 24-48 hr after sun exposure and on bright cloudy days and after fluorescent light exposure. Biopsy of the forehead showed acanthosis with extensive underlying pleomorphic chronic inflammatory cell infiltrate with eosinophils, not extending into the epidermis. It is suggested that this is a case in which a persistent light reactor, whose condition was initiated by exposure to certain chemicals, progressed to actinic reticuloid.

76-2210. Ossa, A. P.; Soto, H. R.; Wolff, F. C.; Armas, M. R. (Dep. Med., Hosp. San Juan de Dios, Santiago, Chile). **Intoxication aguda por talio. [Acute thallium poisoning.]** *Rev. Med. Chile* 103(4): 256-259; 1975. (16 references) (Spanish)

Eight cases are described of acute thallium poisoning following ingestion of rat poison in attempted suicide. In three cases, laboratory determinations of porphyrins and their precursors showed high coproporphyrin levels in two patients and high delta aminolevulinic acid levels in the third. These abnormalities were detected one month after intoxication in the first two cases. The clinical and biochemical similarities between the acute stage of porphyria and thallium poisoning are noteworthy. Although there is no specific therapy, propranolol administration produces improvement of symptoms which are apparently

not related to high catecholamine levels. Similar observations have been made during the critical stage of porphyria, and it is suggested that this adrenergic inhibitor acts upon porphyrin metabolism, which seems to be modified in thallium and other heavy metal poisoning.

76-2211. Popa, G. (Inst. Med. Pharm., Iasi, Romania). **Tratamentul intoxicatiei cu organofosforate. [Treatment of intoxications with organophosphates.]** *Rev. Med. Chir.* 78: 191-194; 1974. (1 reference) (Romanian)

Guidelines for treatment of organophosphate poisoning are presented. Treatment is directed toward the manifest nicotinic and muscarinic symptoms. Confirmation of poison identity is necessary prior to treatment. Continued poison absorption from skin, clothing, or the digestive tract must be prevented. Atropine should be given repeatedly in large doses to just below the point of atropine intoxication, as judged by pulse rate, dryness of the mouth, and mydriasis. Pilocarpine or phenylephrine are given in cases of atropine intoxication. Pralidoxime or obidoxime (Pirangyt) should be administered for a duration determined by clinical status, e.g., persistence of muscular fasciculations and headache. Maintenance of respiratory integrity is imperative. Barbiturates may be required to control convulsions, and correction of the alkali reserve may be necessary. Chronic intoxication may also occur.

76-2212. Fujita, K.; Suzuki, H.; Ochiai, F. (Sch. Ophthalmol., Saitama Med. Coll., Urawa, Saitama, Japan). **[Keratoconjunctivitis due to pesticides of relatively low acute toxicity.]** *Rinsho Ganka (Clin. Ophthalmol.)* 30(4): 419-423; 1976. (10 references) (Japanese)

Twelve cases of keratoconjunctivitis due to pesticides (5 cases due to paraquat dichloride, 2 cases due to 4,5,6,7-tetrachlorophthalide, and one case each due to *o-sec*-butylphenyl *N*-methylcarbamate, pentachloronitrobenzene, blasticidin M, dichlorvos, and an unknown compound, possibly paraquat dichloride) were seen by the authors in the past 4 years. The course and treatment of these cases were reported. Except for blasticidin M (containing organomercury and already banned), no severe general intoxication symptoms were observed. In the cases of paraquat and pentachloronitrobenzene exposure corneal injury appeared in the early stages with more severe symptoms and took longer to reach remission. All cases were resolved within a month without leaving any sequelae. Almost all cases occurred during the application of pesticides, showing the necessity of taking care in pesticides application, even with those of relatively low apparent acute oral toxicity.

76-2213. Nagamatsu, K.; Igata, A. (Third Dep. Internal Med., Fac. Med., Kagoshima Univ., Kagoshima, Japan). **[Arsenical neuropathy. Report of two cases.]** *Rinsho Shinkeigaku (Clin. Neurol.)* 15(1): 1-4; 1975. (9 references) (Japanese)

The accidental ingestion is reported of an arsenical insecticide by three members of a family. A 9 yr old boy died 28 hr after ingestion due to acute arsenical intoxication. A 33 yr old housewife vomited severely during the 28 hr following ingestion. On the third day she noted pigmentation around her mouth which lasted one month. On day 5 she fainted several times. Day 7 showed tingling in both feet which gradually ascended up to both knee joints. Symptoms of paresthesia appeared on the radial sides of both arms. Muscle cramps appeared in both calves. All symptoms progressed until day 50 of hospitalization. No weakness or paresis was noted subjectively during the whole period. On day 55 a neurological examination confirmed no marked abnormal findings except some mild sensory impairment of the distal part of both legs. Motor conduction velocity was prolonged in the median and posterior tibial nerve. A 56 yr old female vomited continuously for 16 hr after ingestion, but did not show any further symptoms. It is concluded that the prolongation of the motor conduction velocity could be considered as a useful electrophysiological finding to confirm the diagnosis of a mild or latent arsenical neuropathy.

76-2214. Fuermaier, B. (Bayer. Landesamt Umweltschutz, Munich, Germany). **Schadlose Beseitigung von Pflanzenbehandlungsmitteln. [Safe disposal of pesticides.]** *Schriftenr. Ver. Wasser Boden Lufthyg. Berlin-Dahlem* 46: 135-138; 1975. (German)

Legal regulations and practical implications are discussed of the safe disposal of pesticides and pesticide-contaminated packaging materials in West Germany. Small quantities of pesticides (in the gram range) can be disposed of together with municipal refuse intended for composting. Otherwise, pesticide wastes should be excluded from the composting process. Except for pesticides containing heavy metals, e.g., cadmium, mercury, and arsenic preparations, pesticides should be disposed of in waste incinerators. Large quantities should be destroyed in special rather than in municipal waste incinerators. Preparations containing heavy metals and arsenic should be disposed of on special landfills, e.g., underground. Unsold preparations and those withdrawn from circulation should be disposed of by returning them to the manufacturer. Contaminated packaging materials should be compacted and disposed of on landfills.

76-2215. Wachendorff, R. (Landeswirtschaftskammer Rheinland-Pflanzenschutz, 5300 Bonn-Bad Godesberg 1, Germany). **Bisherige Ergebnisse bei Aktionen zur Beseitigung von Pflanzenschutzmittel-Resten im Nordrheingebiet. [Results of cases concerning elimination of pesticide residues in the North Rhine area.]** *Schriftenr. Ver. Wasser Boden Lufthyg. Berlin-Dahlem* 46: 139-146; 1975. (German)

Recent legal regulations and experience concerning the disposal of pesticide wastes in the North Rhine area, West Germany, are discussed. The disposal of pes-

ticides on sanitary landfills or dumping is illegal. Such wastes must now be sent to the Emscherbruch central special waste disposal facility at Gelsenkirchen-Resse for incineration or for disposal in iron casks, depending on their hazards. Leftover amounts of spray suspensions and solutions should be distributed on the treated fields, or on barren areas from which they can not run off into water bodies.

76-2216. Trenel, J. (Inst. Wasser-, Boden- und Lufthyg. Bundesgesundheitsamtes, Berlin Germany). **Pruefmodelle aus der Sicht der Wassergefahr und bei Unfaellen waehrend Lagerung und Transport. [Test models for water contamination in case of accidents during storage and transport.]** *Schriftenr. Ver. Wasser Boden Lufthyg. Berlin-Dahlem* 46: 165-174; 1975. (12 references) (German)

Nine different test methods are described for the evaluation of water contamination with toxic substances due to accidents in connection with the storage and transport of toxic and hazardous materials. The tests currently under investigation for possible use in standardization are the A-Z (Assimilation-Consumption) test according to Knoepp, the bacterial toximetric test according to Axt, Bringmann's test on the incipient inhibition of glucose assimilation by *Pseudomonas*, the peptone degradation inhibition test with Sepromat according to Offhaus, the determination of the minimal growth inhibition concentration according to Schubert, the oxygen consumption test according to Robra, Bringmann's test for the determination of the biological effect of hazardous and toxic substances on algae, fish toxicity test with orfe, and the subjective determination of the odor threshold concentration.

76-2217. Rubtsov, A. F. (Inst. Forensic Med., USSR Ministry of Health, Moscow, USSR). **Smertel'nye otravleniya v 1972-1973 gg. [Fatal poisonings in 1972-1973.]** *Sud.-Med. Ekspert.* 18(4): 24-26; 1975. (Russian)

Statistical data is presented on fatal poisonings in the USSR between 1972 and 1973. Pesticides accounted for 3.156% of all fatal poisonings in 1972, and for 3.445% in 1973. Organophosphorus pesticides were responsible for 2.829% of all poisonings in 1972, and for 2.992% in 1973. Compared with the 1970-1971 level, the incidence of fatal poisonings with pesticides increased by 0.3%. No absolute values are given.

76-2218. Budrin, Yu. P.; Meshcherskaya, K. A. (Dep. Forensic Med., Vladivostok Med. Inst., Vladivostok, USSR). **Otravleniya soedineniyami talliya. [Intoxications with thallium compounds.]** *Sud.-Med. Ekspert.* 18(4): 37-39; 1975. (Russian)

Two cases are reported of acute poisoning due to involuntary ingestion of thallium compounds. Asthenia, tremors, numbness of the limbs, headache, emesis, hyperesthesia with hyperemia, disturbances in coordination and orientation, involuntary urination, oral automatism, and disturbances in sleep were observed in a 32-yr-old man who had ingested 80 ml of 1.15% solution of thallium nitrate. A urine thallium level of 3.6 g% and a serum thallium level of 1.4 g% were measured more than one month after poisoning. The patient was discharged from the hospital in satisfactory condition. Another 25-yr-old woman developed sharp pains in the lower limbs, polyneuritis, microhematuria, and cylindruria after ingestion of a reagent containing thallium formate and malonate. She died in cardiovascular insufficiency. Inflammation of the intestinal mucosa, ecchymosis and edema of the myocardium, atrophy of the skin and subcutaneous tissue, dystrophy and degeneration of the parenchymatous organs, multiple diapedetic hemorrhage with focal glial proliferation in the brain, isolated perivascular lymphocyte infiltrations, dystrophic changes in neurons, metaplasia of the bronchial epithelium, and interstitial myocarditis were found on autopsy.

76-2219. Graul, E. H. (Klin. Poliklin. Nuklearmed., Radiologiezentrum der Phillips-Universitaet, Marburg/Lahn, Germany). **Umwelt und Krebsentstehung. [The environment and carcinogenesis.]** *Therapiewoche* 26(6): 743-761; 1976. (German)

Physical, chemical, and viral carcinogenic factors in the environment, and the biochemical principles of cell transformation under the action of carcinogens are reviewed. Insecticides, fungicides, and bactericides containing dialkylnitrosamines, *N*-nitroso cyclic amines, and *N*-alkyl-*N*-arylnitrosamines as active agents have been homologated in the last 15 years in the USA, England, and West Germany.

76-2220. Brooker, M. P.; Edwards, R. W. (Dep. Appl. Biol., Univ. Cambridge, Cambridge, England). **Aquatic herbicides and the control of water weeds.** *Water Res.* 9(1): 1-14; 1975. (126 references)

Evidence is examined for the actual and potential effects of herbicide use, particularly where such effects constrain other water-resource uses or differ from those resulting from alternative weed control methods, principally cutting and subsequent removal. The review covers specific areas of herbicides used in fresh water; classification of effects of herbicide application; direct effects on potable water supplies, industrial water supplies, irrigation and farmstock; as well as direct effects on aquatic non-target organisms. Indirect effects on the oxygen-carbon dioxide balance, plant replacement, and the effects of plant death on the aquatic fauna are also considered.

76-2221. Suchomski, T. J. (Arbeitshyg.-Abt. Slupsk, 76-200 Poland). **Die Epidemiologie der Beruefs- und akzidentellen Intoxikationen durch Pflanzenschutzmittel in einer agro-industriellen Woiwodschaft. [Epidemiology of occupational and accidental pesticide intoxications in an agro-industrial center.]** *Z. Gesamte Hyg.* 22(2): 91-92; 1976. (German)

Cases of occupational and accidental poisonings with pesticides in an agroindustrial province of Poland during the 1963-1973 period are analyzed. Occupational poisonings were reported in 48 cases, and there were 26 cases of accidental poisoning, including two with lethal outcome, during the 1966-1973 period. Occupational poisonings were mainly due to organophosphorus compounds, and occurred most frequently among young male agricultural workers (tractor drivers, farmhands, material handlers) without sufficient working experience. Occupational poisonings with pesticides accounted for 28% of all industrial poisonings. Occupational poisonings were usually milder than accidental ones.

76-2222. Smerling, M. (Inst. Rechtsmed., Freien Univ. Berlin, D-1000 Berlin 33, Germany). **Ein Fall von Ueberlebter Giftbeibringung (E 605 forte). Rechtsmedizinische, klinische, toxikologische und juristische Aspekte. [Forensic and clinical toxicologic aspects in a nonfatal case of criminal poisoning with E 605 forte.]** *Z. Rechtsmed.* 77(2): 149-155; 1976. (26 references) (German)

Clinical, toxicological, and legal aspects are described of the criminal poisoning of a 60-year-old man with E 605 forte by his wife. Bradycardia, hypersecretion, spasm of the muscles of the limbs, diarrhea, and miosis were the first symptoms of the poisoning after the intake of poisoned food. Cessation of spontaneous respiration and atrial fibrillation developed during therapy for an alleged heart attack. Repeated i.v. atropinization had a surprisingly good therapeutic effect. The patient survived without any complications. The quantity of the poison ingested is tentatively estimated at 150 mg. It reached or slightly exceeded the individual lethal dose.

76-2223. Poehlmann, E.; Schwerd, W. (Inst. Rechtsmed., Univ. Wuerzburg, D-8700 Wuerzburg, Germany). **Zum Nachweis von E 605 in einer nach 21monatiger Liegezeit exhumierten Leiche. [Detection of parathion (E605) in a corpse exhumed after 21 months.]** *Z. Rechtsmed.* 77(3): 233-236; 1976. (7 references) (German)

The recent poisoning of the 52-year-old father-in-law of a dental assistant with parathion-containing pralines prompted exhumation of the suspect's father 21 months after he died of an alleged heart attack. Gastric and intestinal contents were subjected to steam distillation and extraction with ether. A cholinesterase-inhibiting fraction was isolated by thin-layer chromatography according to the Geldmacher-Von Mallinckrodt modification of Ackermann's method. Parathion was detected by palladium chloride. Ethyl chloride was detected gas chromatographically according to Machbert's method. A biological test with *Drosophila melanogaster* was positive. The dose of parathion ingested was slightly higher than the individual lethal dose. The suspect admitted adding parathion into the tea her father drank shortly before he died.

76-2224. Kokordak, J.; Kacmar, P. (Natl. Inst. Vet. Med., Kosice, Czechoslovakia). **Praped hromadnej otravy ryb endrinom-20. [Mass intoxication of fish by endrin-20.]** *Vodni Hospod.* 24(6): 163-165; 1974. (3 references) (Slovak)

The effects of the pesticide endrin-20 were observed on the biocenosis of the Kanas Reservoir and on carp and other plant-eating fish. Carp were cultivated in this reservoir for food sources and for sport. When endrin-20 was used near inlets for control of mice, the biocenosis of the reservoir was seriously disturbed and 100% of the fish died, presumably due to endrin from intoxication. The nervous system appeared to be affected before other organs. Endrin-20 is very toxic and it is recommended that standards relevant to the locality of application be followed.

76-2225. Horiguchi, Y.; Yanagisawa, T.; Sasaki, K.; Ide, H.; Ueki, S.; Abe, E.; Kurosawa, K. (Jpn. Inst. Rural Med., Saku Gen. Hosp., Usuda, Nagano, Japan). **[Neurological studies on the treatment of organophosphorus insecticide intoxication. Part 5. Electroencephalographic study of pesticide applicators.]** *Noyaku No Seitai Eikyo Ni Kansuru Kenkyu (Studies on the Effects of Pesticides on Living Bodies)* 1976, p. 68-70. (Japanese)

Farmers with more than 10 yr experience in organophosphorus insecticide application were subjects of a 1974-75 neurological examination. The pesticide applications were made in vinyl greenhouses. Results showed a tendency toward retardation of the basic EEG rhythm during arousal and at rest, even before the application season. Re-examination in the current year showed the same slow alpha activity before application (58% in men and 8% in women). The post-application EEGs were nearly the same as the pre-application EEGs. The ChE activity in serum and erythrocytes was lower than the standard range before application, and post-application values showed a tendency towards reduction.

76-2226. Obihiro City Office. (Hokkaido, Japan). **[Environmental pollution due to pesticides.]** *Obihiro-Shi Shimin Kotsu Kogai-Ka (Current Status of Environmental Pollution in Obihiro City)* No. 6, 1974, p. 21. (Japanese)

Because of the kinds of crops grown and their primary cultivation under upland conditions, cases of pesticide residues detection on agricultural crops were rare in the Tokachi area of Hokkaido. However, due to the excessive application of pesticides and the practice of washing containers with river water, fish deaths were numerous. In 1974, three eposides of river fish kills occurred, presumably due to pesticides. Salmon and trout died just after stocking into stream, and dead rainbow trout were found in a fish farm into which pesticide contaminated water ran. Environmental (air and water) pollution caused by improper disposal of empty pesticide containers or by burning such containers should not be overlooked.

76-2227. Rieger, H.; Okonek, S. (Neuro-Psychiatr. Med. Klin., Johannes-Gutenberg Univ., Mainz, Germany). **L'E.E.G. dans les intoxications par les inhibiteurs de la cholinesterase (insecticides organo-phosphores). [The EEG in intoxications by cholinesterase inhibitors (organophosphorus insecticides).** *Rev. Electroencaphalogr. Neurophysiol. Clin.* 5(1): 98-101; 1975. (13 references) (French)

EEGs were recorded in 17 cases of organophosphate intoxication and correlated with the acetylcholine level, concentration of toxic substance, serum cholinesterase activity, and clinical symptoms. In the initial acute stage, the patient is in a coma with a fairly rapid, low, or average amplitude rhythmic EEG activity for about 3 days. Levels of acetylcholine and the toxic substance are at their peaks, and there is no measurable cholinesterase activity. In the following stage, the EEG is clearly more synchronous and of high amplitude. The patient is in a coma and either areactive or slightly reactive to very strong stimulation. Acetylcholine and the toxic substance have decreased considerably, but cholinesterase activity is still not detectable. In the third stage, cholinesterase activity rises again and rapidly exceeds the critical limit of 20-30%. The patient is now in a state of somnolence. A labile alpha rhythm reappears in the EEG. Muscular response begins to recover, indicating that the permanent depolarization caused by acetylcholine has been overcome. From then on, the EEG may still display an excess of diffuse or temporal theta waves for weeks before the alpha waves stabilize. Use of the EEG can differentiate acute insecticide poisoning from barbiturate intoxication. In insecticide poisoning the wave pattern is more or less desynchronized and has a residual areactive activity of alpha and beta frequency, whereas with barbiturates there is predominant delta activity, possibly associated with periodic gaps and bursts.

see also **76-2245**

76-2228. Zimmering, S. (Div. Biol. Med. Sci., Brown Univ., Providence, RI 02912). **Utility of *Drosophila* for detection of potential environmental chemical mutagens.** *Ann. N.Y. Acad. Sci.* 269: 26-33; 1976. (52 references)

Drosophila offers the quickest and most efficient in vivo animal system available today for genetic detection of transmitted mutations and chromosomal aberrations induced in germ line cells by environmental agents. It can also detect genetic damage induced by mutagens that require activation by liver microsomal enzyme systems. Various screening tests are commonly used to reveal: deleterious recessive gene or point mutations and small deficiencies induced in germ line cells of the male; reciprocal translocations involving the Y chromosome and chromosomes 2 and 3; loss of the X or Y chromosomes, chromosome 4, and parts of the Y chromosome due to breakage effects in postmeiotic cells; and nondisjunction of the sex chromosomes and the small chromosome 4. *Drosophila* can be used to detect mutagenic activity in tissues and body fluids of treated rodents, which suggests the possibility of its utilization as in indirect indicator to detect mutagenic activity in human body fluids. *Drosophila* might also be used as a test system for monitoring the immediate environment, especially of workers in potentially high risk industries.

76-2229. Valencia, R. A. (Mutagenesis Lab., WARF Inst., Inc. Madison, WI 53701). **The use of *Drosophila* for mutagenesis screening.** *Ann. N.Y. Acad. Sci.* 269: 34-36; 1975.

Mutagenesis screening with *Drosophila* can be performed on a large scale in an efficient and effective manner. Only one trained and experienced person is needed in any particular testing laboratory to plan, initiate, oversee, and interpret testing, and flies can be exposed outside the laboratory, then brought in for genetic tests. The Mutagenesis Laboratory of WARF Institute, Inc. is geared to screen for three types of genetic end-points: dominant lethals, chromosome breakage, and rearrangement; nondisjunction and loss; and sex-linked recessive lethals. A selective screening scheme for chromosome damage is also employed. To deal effectively with weakly mutagenic chemicals, treated and control males are mated individually, the F_1 female matings from each male are kept grouped, and each male is transferred sequentially through four broods in order to sample the entire range of germ cell developmental stages. The search continues for new and/or improved procedures, keeping in mind the special circumstances of mutagen screening. A pesticide (not specified) is used as an example of low-level mutagen that may produce erroneous conclusions. The pesticide yielded 27 lethals in 2803 tests, for a frequency of 0.963%, high compared to the control frequencies of 0.14%. The current test method showed that 21 of the 27 mutations were from a single male in all stages of gametogenesis, and therefore probably resulted from a spontaneous mutation. The pesticide was probably not mutagenic.

76-2230. Chapman, P. J. (Dep. Biochem., Univ. Minnesota, St. Paul, MN 55108). **Microbial degradation of halogenated compounds.** *Biochem. Soc. Trans.* 4(3): 463-466; 1976. (36 references)

The degree of persistence of organochlorine compounds increases with the degree of chlorine substitution in such molecules, higher chlorinated compounds are not readily degraded by microorganisms. When this biodegradation slows down or is prevented, accumulation of the compounds occurs within the environment, with subsequent possible concentration and redistribution through the tissues of organisms constituting food chains. Bacteria and fungi are the main agents for returning organic carbon compounds to the carbon cycle. The organisms which effect mineralization of naturally occurring organochlorine compounds are also expected to degrade those of synthetic origin. However, when chemicals of synthetic origin represent novel additions to the environment, their degradation will occur only if they are substrates for preexisting bacterial enzyme systems or after bacteria have evolved the necessary enzymes for their degradation. Persistence of some synthetic products in soil and water may therefore reflect the fact that insufficient time has elapsed for evolution of competent biochemical mechanisms. It is not unexpected that bacteria may do little more than modify the structures of persistent organochlorine chemicals. These chemicals are sufficiently different from those found naturally so that extensive degradation by specific or fortuitous reaction sequences is precluded.

76-2231. Grant, B. F. (U.S. Fish Wildlife Service, Southeastern Fish Cultural Lab., Marion, AL 36756). **Endrin toxicity and distribution in freshwater: a review.** *Bull. Environ. Contam. Toxicol.* 15(3): 283-290; 1976. (49 references)

Endrin is one of the most toxic of all economic poisons to fish, and is slightly less toxic to freshwater insects and crustacea. The effects of endrin are noncumulative and the symptoms at the onset of intoxication are reversible. Chronic dietary exposure in fish affects growth, gonad development, thyroid activity, serum characteristics, body fat, behavior, thyroid cell size, gametogenesis, and liver lipids. The pesticide is rapidly accumulated in body tissues from contaminated food and water; tissue levels in fish are proportional to amounts in food, and blood endrin levels are directly correlated with lethal thresholds after exposure. Algae can also concentrate endrin manyfold. Genetically based endrin resistance in populations of fish from areas of heavy endrin contamination is reported, and feeding experiments with resistant fish demonstrate potential hazards of accumulating residues. Suspended particles of activated carbon increased endrin toxicity to fish, and mud accumulates large quantities of the pesticide, although it does not release it into standing water. Endrin is extremely resistant to degradation. Endrin pollution of fresh water has been reported extensively over the past decade; residues in water and biota reflect seasonal agricultural use of pesticides and irrigation water.

76-2232. Folmar, L. C. (Fish-Pesticide Res. Lab., Denver, CO). **Overt avoidance reaction of rainbow trout fry to nine herbicides.** *Bull. Environ. Contam. Toxicol.* 15(5): 509-514; 1976. (17 references)

Rainbow trout fry (*Salmo gairdneri*) were placed in a maze apparatus in which one arm contained untreated water and the other arm contained water treated with various concentrations of 2,4-D, Aquathol K (endothall), copper sulfate, dalapon, diquat, xylene, trichloroacetic acid (TCA), acrolein, or glyphosate. The number of fish in each arm was determined after 1 hr. The fry avoided copper sulfate (0.0001 mg/l), dalapon (1.0 mg/l), 2,4-D, (1.0 mg/l), xylene (0.1 mg/l), and acrolein (0.1 mg/l), but did not avoid glyphosate, Aquathol K, or diquat at concentrations below 10 mg/l or TCA at concentrations less than 1.0 mg/l. The fish showed a significant attraction to xylene at a concentration of 0.01 mg/l, but avoided a concentration of 0.1 mg/l. The levels of avoidance demonstrated by the test organisms differed widely according to the herbicide tested. The results suggest that rainbow trout would not be killed by, and would not avoid, relatively low concentrations of dalapon, 2,4-D, glyphosate, Aquathol K, diquat, or TCA. Trout would not avoid a lethal exposure to acrolein, but they would avoid nontoxic concentrations of copper sulfate and xylene.

76-2233. Dikshith, T. S. S.; Datta, K. K.; Chandra, P. (Ind. Toxicol. Res. Cent., Lucknow, India). **90 Day dermal toxicity of DDVP in male rats.** *Bull. Environ. Contam. Toxicol.* 15(5): 574-580; 1976. (19 references)

DDVP (dichlorvos, 21.4 mg/kg) in 0.5 ml ethanol was applied 5 days/wk for 13 wk to the shaved lateroabdominal area of male albino rats. Sections of skin and testis taken at intervals of 7-90 days after the commencement of treatment were examined histologically. None of the animals showed symptoms of insecticide poisoning or macroscopic evidence of dermatitis or other skin lesions. Microscopic examination of the treated skin showed no pathological changes, although certain cellular changes (vacuolization of the epidermal cells and thinning of the epidermis) were more marked in the treated epidermis than in the untreated control skin after 90 days. Histological examination of the testis also showed no consistent damage, although necrosis of the majority of the seminiferous tubules was apparent in two experimental rats and one control rat. Rapid degradation of DDVP and detoxification in the liver seem to be the prime reasons for its lack of toxicity in the rat.

76-2234. Schaper, R. A.; Crowder, L. A. (Dep. Entomol., Univ. Arizona, Tucson, AZ 85721). **Uptake of ^{36}Cl-toxaphene in mosquito fish, *Gambusia affinis*.** *Bull. Environ. Contam. Toxicol.* 15(5): 581-587; 1976. (5 references)

Mosquitofish (*Gambusia affinis*) were exposed for up to 10 hr to aged tap water containing 2 ppm ^{36}Cl-toxaphene in acetone; in some cases, the fish were then transferred to aquaria containing untreated water. Tissue pesticide residues were determined by liquid scintillation counting. The uptake of ^{36}Cl-toxaphene and toxaphene-related (TR) residues was a linear function of and directly proportional to the length of exposure. There was no indication of excretion of toxaphene or TR residues during the first 6 hr following exposure. Also, metabolic alteration of toxaphene during the experiment appeared to be minimal. The toxicity syndrome exhibited by the intoxicated fish was divided into five stages. By the time the fish exhibited the first toxicity response to the pesticide (stage 2), they had sorbed 90.3% of the average fatal residue. Fish characterized as normal (stage 1) had accumulated 35% of the fatal residue. The onset of specific toxicity symptoms was directly proportional to the pesticide body load. Thus, differences in the toxicity response to toxaphene by individual fish appeared to be due to different body loads rather than to different tolerances to a particular toxicant level.

76-2235. Saxena, S. C.; Karel, A. K. (Toxicol. Lab., Dep. Zool., Univ. Rajasthan, Jaipur, India). **On effect of chlordane on the blood glucose and of glucose administration of the acute chlordane toxicity in *Meriones hurrianae* Jerdon, the Indian desert gerbil.** *Bull. Environ. Contam. Toxicol.* 15(5): 593-600; 1976. (16 references)

Chlordane (0.2 cc of a 1.0-3.0% petroleum solution) was administered i.m. to healthy adult male gerbils (*Meriones hurrianae* Jerdon), after which, in some cases, 2 g/kg of a 50% glucose solution was administered orally. Blood glucose levels were determined at intervals of 45 min to 14 days after treatment. Chlordane administration alone caused hyperglycemia in the treated animals; the maximum blood glucose level was reached 1 hr after treatment. The blood glucose level gradually returned to control values within 1 wk. The chlordane effect on blood glucose was dose-dependent. In animals given oral glucose, chlordane decreased glucose tolerance. The results suggest that chlordane may stimulate glucagon secretion and inhibit the synthesis of glucose, possibly via destruction of cells of the islets of Langerhans. Chlordane also appears to impair the uptake and utilization of glucose in the intoxicated gerbil.

76-2236. Vance, B. D.; Maki, A. W. (Dep. Biol. Sci., North Texas State Univ., Denton, TX 76203). **Bioconcentration of Dibrom by *Stigeoclonium pachydermum*.** *Bull. Environ. Contam. Toxicol.* 15(5): 601-607; 1976. (17 references)

Unialgal cultures of *Stigeoclonium pachydermum*, a common green alga, were inoculated with 0.50 μg/ml of Dibrom (naled) dissolved in acetone. Growth was measured for 6 days after inoculation, and pesticide residues in the cultures were determined by gas chromatography. Concentrations of Dibrom exceeding 20 μg/ml were required to inhibit the growth of *S. pachydermum*. The treatments caused the formation of a thick amorphous

coating on the filaments and loss of the characteristic setae at the branch tips. Bioconcentration of Dibrom by the algae appeared to be accomplished via adsorption of the insecticide to the cell wall rather than by absorption into the cells. The results suggest a chemical reaction between Dibrom and the algal cell wall during which the pesticide is rapidly degraded.

76-2237. Nelson, J. H.; Stoneburner, D. L.; Evans, E. S., Jr.; Pennington, N. E.; Meisch, M. V. (U.S. Army Environ. Hygiene Agency, Aberdeen Proving Ground, MD 21010). **Diatom diversity as a function of insecticidal treatment with a controlled-release formulation of chlorpyrifos.** *Bull. Environ. Contam. Toxicol.* 15(5): 630-634; 1976. (5 references)

Cultivated rice fields were treated with a chlorinated polyethylene pelletized formulation of 10.6% chlorpyrifos to produce a level of 0.25-2.0 ppm in water based on a theoretical total initial release of all active ingredients. Randomly selected treatments were replicated three times. Diatometers placed in the water covering the fields were analyzed 6 and 12 wk post-treatment. At 6 wk post-treatment, the chlorpyrifos-treated and untreated control plots did not differ substantially with respect to diatom diversity. However, in all treated fields, diatom diversity decreased significantly between 6 and 12 wk post-treatment, whereas diversity increased in the control fields. At 12 wk, estimates of mean diversity were greatest for the control fields, followed in decreasing order by those treated with 0.25, 0.5, 1.0, and 2.0 ppm. No increase in the degradation rate of chlorpyrifos was observed over the 12-wk period, and the diatom population had no noticeable effect upon the larvicidal activity of the insecticide.

76-2238. Sharma, P. R. (Dep. Molecular Biol., Univ. Geneva, Sci. II, Geneva, Switzerland). **Inhibition of some microbial functions of soil by the fungicide phenylmercuric acetate.** *C. R. Soc. Phys. Hist. Nat. Geneve* 10(2/3): 170-175; 1975. (20 references)

Air-dried sandy-loam farm soil was treated with 300 ppm ammonium sulfate and 80-240 ppm phenylmercuric acetate (PMA) in water. The moisture of the soil was adjusted to 40% of saturation capacity and determinations were made of bacterial numbers, nitrification, and respiration. At 0.16 ppm, PMA had no effect on any of the parameters tested, but at 80-250 ppm, it inhibited all three microbial functions. Total bacterial counts and soil respiration remained almost static for the first 14 days after treatment with 240 ppm, and thereafter they started increasing. Soil nitrification was inhibited to a lesser degree.

76-2239. Lijinsky, W.; Taylor, H. W. (Carcinogenesis Program, Biol. Div., Oak Ridge Natl. Lab., Oak Ridge, TN 37830). **Carcinogenesis in Sprague-Dawley rats of *N*-nitroso-*N*-alkylcarbamate esters.** *Cancer Lett.* 1(5): 275-279; 1976. (4 references)

The effects of nitrosocarbaryl, nitroso-*N*-methylurethane, and nitroso-*N*-ethylurethane administered by gavage in olive oil solution to groups of 12 female Sprague-Dawley rats were studied. The rats received 0.2 ml of 0.11 *M* solution once a week for 10 weeks, for a total dose of 0.22 mmol. Male rats received 0.2 ml of a 0.16 *M* solution for 20 weeks. Most tumors were produced in the non-glandular part of the stomach. Metastases to liver and lung and serosal implantations were seen in all groups. In females, nitrosomethylurethane and nitrosoethylurethane produced similar survival patterns. All 12 animals receiving nitrosoethylurethane died with invasive squamous cell carcinoma of the stomach; 5 of the 12 receiving nitrosomethylurethane died with this tumor, 3 had papillomas of the stomach,and 1 had a squamous cell carcinoma in the nasal turbinates. Animals given nitrosocarbaryl lived longer than those given the ethyl esters. Males receiving the higher dose of nitrosocarbaryl died earlier than the females receiving a lower dose. Nitrosocarbaryl is a sufficiently potent carcinogen to suggest that its formation in nature or *in vivo* from carbaryl residues in food and nitrite might represent a significant source of carcinogen for humans.

76-2240. Eisenbrand, G.; Schmahl, D.; Preussmann, R. (Inst. Toxicol. Chemother., German Cancer Res. Cent., D-6900, Heidelberg, Germany). **Carcinogenicity in rats of high oral doses of *N*-nitrosocarbaryl, a nitrosated pesticide.** *Cancer Lett.* 1(5): 281-284; 1976. (4 references)

The effects of high doses (130 mg/kg) of *N*-nitrosocarbaryl administered p.o. to 31 male Sprague-Dawley rats were studied. Of the treated animals, 9 developed squamous cell carcinomas in the forestomach, 2 had papillomas,and 6 had hyperkeratoses, also in the forestomach. Tumors were not observed in other organs. The average time of death of animals with carcinoma was 167 days from the start of the experiment. The average total dose applied was about 5000 mg/kg. Early deaths were also recorded among animals with hyperkeratoses and papillomas. It is suggested that these lesions were indicative of an early stage of the carcinogenic effect of this compound. At the time when the incidence of tumors was strongly increasing (after day 200) most of the treated rats were already dead. This clearly shows that the dosage was so high that the greater proportion of the treated rats died from cumulative toxic effects of *N*-nitrosocarbaryl rather than from tumors caused by the compound. This report confirms that *N*-nitrosocarbaryl is a locally acting potent carcinogen when administered p.o. in the rat and confirms earlier reports of local sarcomas resulting from s.c. injection of single doses.

76-2241. Kuhr, R. (Dep. Entomol., New York State Agric. Exp. Stn., Geneva, NY 14456). **Insecticide metabolites in and on plants.** *Chem. Tech.* 6(5): 316-321; 1976. (13 references)

The major metabolic pathways of the or-

ganochlorine, carbamate, and organophosphorus insecticides are discussed in plants. Plants are capable of dehydrochlorination and reductive dechlorination of organochlorines and additional unknown water-soluble products have been found. The majority of carbamates are subject to hydrolysis in biological systems, although carbamates in plants are more often subject to oxidation in the form of hydroxylation, epoxidation, dealkylation, and sulfoxidation. Metabolites from both hydrolysis and oxidation are subject to conjugation in plants. Hydrolysis to nontoxic metabolites is also an important degradation route in the case of the organophosphorus insecticides, as are oxidation and conjugation. Surface residues of carbamate and organophoshorus insecticides are degraded by air and light to a wide variety of products. The significance of the various insecticide metabolites depends on their toxicity, particularly to mammals, the amounts present, and the fate of the residues during food processing. Significance must also be balanced against economy and need.

76-2242. Wiener, M.; Pittman, K. A.; Stein, V. (Inst. Comp. Human Toxicol., Albany Med. Coll., Albany, NY 12208). **Mirex kinetics in the Rhesus monkey. I. Disposition and excretion.** *Drug Metab. Dispos.* 4(3): 281-287; 1976. (17 references)

A study was undertaken of the disposition and excretion of mirex in a primate, with a second purpose of accumulating data required for a pharmacokinetic study of mirex disposition. Three female rhesus monkeys received ^{14}C-mirex (5.23 mCi/mmol) i.m. (in 2) or p.o. (in 1) at a dose of about 1 mg/kg. Following i.v. administration, plasma ^{14}C showed a rapid decrease over the first few hours from the initial high levels. In the monkey receiving the compound p.o., ^{14}C first appeared in the plasma at 2 hr and reached a maximum at 5 hr. After 2 weeks the rate of decline in all animals was very slow. Excretion and tissue distribution were essentially the same regardless of the method of administration. Less than 0.6% of the dose was found in the urine. ^{14}C was excreted in the feces for the duration of the experiment with a maximum cumulative excretion of 7% after 388 days. At autopsy all tissues analyzed contained ^{14}C. The highest concentration of ^{14}C was in the fat (about 80% of the total dose), followed by the adrenal, peripheral nerve, thyroid, and skin. At least 95% of the radioactivity present in the fat and feces was unchanged mirex. A small amount of a compound more polar than mirex was noted in the feces, accounting for less than 3% of the fecal radioactivity.

76-2243. Pittman, K. A.; Wiener, M.; Treble, D. H. (Inst. Comp. Human Toxicol., Dep. Biochem., Albany Med. Coll., Albany, NY 12208). **Mirex kinetics in the Rhesus monkey. II. Pharmacokinetic model.** *Drug Metab. Dispos.* 4(3): 288-295; 1976. (30 references)

A study was initiated to develop a kinetic model of the fate of mirex in the Rhesus monkey. ^{14}C-Mirex was administered i.v. and p.o. to female Rhesus monkeys in doses ranging between 0.861 and 1.05 mg/kg. Radioactivity was measured in plasma, urine, and feces at intervals during the study and in tissues after sacrifice. To provide estimates of the values of the first-order rate constants required by the proposed pharmacokinetic models, graphical analysis of plots of the logarithm of plasma concentration vs. time was used. A BASIC-language program, FITKIN, was used to obtain numerical solutions to the differential equations for each model and to adjust the estimates in order to obtain a normalized, least-squares fit. Of the several models postulated, a mammillary, four-compartment, open-system model, providing for the urinary excretion of mirex from a "central" compartment and for the fecal excretion of mirex from a "fast" compartment, yielded theoretical data in agreement with observed values. This model predicts that the accumulation of mirex into fat would be retarded by the presence of a "slow" tissue compartment, so that distribution equilibrium would take about half a year. From that time to the end of a 5 yr projection little decline in the quantities of mirex would be predicted for any compartment. The sequestration in fat and a lack of metabolism were responsible for the long biological half-life of mirex in the Rhesus monkey.

76-2244. Farkas, I.; Desi, I.; Krausz, T.; Revesz, T.; Kucsera, K. (Orszagos Kozegeszsegugyi Intezet, Budapest, Hungary). **Egy klorozott szenhidrogen, a Lindan, parenteralis es intracysternalis adagolasanak akut hatasa kiserleti allatok agyi elektromos tevekenysegere. [Acute effect of parenteral and intracisternal lindane, a chlorinated hydrocarbon, on the basis electrical activity of experimental animals.]** *Egeszsegtudomany* 20: 65-73; 1976. (18 references) (Hungarian)

EEG changes were registered in rats and rabbits following the oral and intracisternal administration of lindane. An oral LD50 dosage also increases the spontaneous spasm activity during the spasm-free periods of electrical brain activity. The introduction of lindane via a cannula directly into the CSF space induced identical EEG changes; 50 mg/kg per os produced the same effect as a 0.1 mg/g wet brain weight intracisternal dosage. Rabbits were twice as sensitive to the EEG effects of lindane as were the rats. Stroboscopic stress aggravated the EEG changes in both species. The lindane dosage which impairs nervous system activity is several hundred times greater than that affecting human beings exposed to this agent.

76-2245. Ruzicska, P.; Sandor, P.; Jozsef, L.; Endre, C. (Orszago Kozegeszsegugyi Intezet, Chinoin Gyogyszer—es Vegyeszeti Termekek Gyara Rts., Budapest, Hungary). **A Fundazol 50 WP kromoszoma mutagenitasanak vizsgalata. [Study on the chromosomal mutagenicity of Fundazol 50 WP.]** *Egeszsegtudomany* 20: 74-83; 1976. (21 references) (Hungarian)

The effects of Fundazol 50 WP (benomyl) on chromosomal mutagenesis were studied in vitro and in

vivo. Blood cultures from workers producing the compound were also subjected to chromosome analysis. At the dosages tested (25-500 mg/kg/day) Fundazol 50 WP had no chromosomal mutagenic effects on mature Charles River rats. However, the rate of chromosomal aberrations was considerably increased in fetal rat tissues treated with 200-500 mg/kg/day. The human chromosome analyses showed no mutagenic effects, indicating that Fundazol is not injurious to human health in this respect.

76-2246. Torchinsky, A. M.; Orlova, N. V.; Smirnova, E. V.; Makarova, L. F. (Inst. Nutr., Acad. Med. Sci., Moscow, USSR). **Materialy k toksicheskogo-gigienicheskoi kharakteristike pestitsida iz gruppy karbamatov-benlata. [Data on the toxico-hygienic characteristics of the pesticide benlate from the carbamate group.]** *Gig. Sanit.* (2): 35-39; 1976. (Russian)

The LD50 of benlate (benomyl) for rats was found to exceed 3.6 g/kg when administered per os, and the cumulative effects of the compound are weak. The testicles were the most severely affected organs following chronic benlate treatment. The threshold level for toxic action was 62.5 mg/kg in a study where male Wistar rats were treated over a 12 mo period; 12.5 mg/kg was without toxic effect. Determination of benlate residues in vegetables and fruits treated with the pesticide showed them to be within the 1 mg/kg level. It is suggested that benlate residue levels in vegetables and fruits not exceed 1 mg/kg.

76-2247. Engst, R.; Macholz, R. M.; Kujawa, M.; Lewerenz, H. J.; Plass, R. (Cent. Inst. Nutr., Acad. Sci. GDR, GDR-1505 Potsdam-Rehbrucke, Germany). **The metabolism of lindane and its metabolites gamma-2,3,4,5,6-pentachlorocyclohexane, pentachlorobenzene, and pentachlorophenol in rats and the pathways of lindane metabolism.** *J. Environ. Sci. Health Bll* (2): 95-117; 1976. (17 references)

The metabolism of lindane and its metabolites, γ-PCCH, pentachlorobenzene, and PCP, has been studied in rats. Urine, feces, blood, and important organs were analyzed for free metabolites and those bound as β-glucuronides. The results of the study show that lindane is degraded via the intermediates γ-PCCH and two polychlorocyclohexenols, mainly to pentachlorophenol and tetrachlorophenol. While the first degradation step equals a detoxification of 1:20, the second step represents a toxification of 40:1. In the next steps the toxicity always decreases in the ratio 1:2. Detoxification is caused to a great extent by glucuronidation, conjugation to sulfuric acid, and SH-conjugation. The metabolic processes of lindane take place in all organisms according to the same general scheme. For microorganisms there is a greater variety in the possible reactions.

76-2248. Barna, J.; Simon, G. (Kozponti Elelmiszeripari Kutato Intezet, Budapest, Hungary). **Kis-dosisu Birlane etetes hatasa a belfelszivodasra. [Effect of small oral doses of Birlane (chlorfenvinphos) on intestinal resorption.]** *Kiserl. Orvostud.* 26(6): 605-609; 1974. (14 references) (Hungarian)

General physiological changes and changes in the resorption of nutrients through a duodenojejunal section were studied in female albino Wistar rats fed a diet containing 10 ppm of Birlane (chlorfenvinphos) for 30 days, or 30 ppm for 10 days. The statistically insignificant reduction of weight gain was due to a reduced food intake, probably as a result of the impaired organoleptic properties of the contaminated food. The food utilization coefficient decreased by 9% and 16%, respectively, in the two groups. Chlorfenvinphos caused no change in the intestinal resorption of methionine and Ca, the resorption of ^{24}Na showed a slight but significant decrease, and the glucose resorption increased insignificantly in the group fed a diet with 30 ppm of chlorfenvinphos. The serum cholinesterase activity was inhibited in correlation with the pesticide dose administered, and slight inhibition of the erythrocyte acetylcholinesterase activity was observed (the rate of inhibition was 30% in the group fed a diet with 30 ppm of chlorfenvinphos). The rate of inhibition of the cholinesterase activity of the intestinal epithelium was moderate and was about the same in both groups. Autopsy revealed no morphological differences between the two groups.

76-2249. Green, M. H. L.; Muriel, W. J.; Bridges, B. A. (MRC Cell Mutat. Unit, Univ. Sussex, Falmer, Brighton BN1 9QG, England). **Use of a simplified fluctuation test to detect low levels of mutagens.** *Mutat. Res.* 38(1): 33-41; 1976. (10 references)

A modification of the Luria and Delbruck fluctuation test was found to be attractively simple and sensitive as a test for low levels of mutagens or weak mutagens. The test calls for the scoring by eye of individual tubes for the presence or absence of a mutation. This procedure can detect concentrations of mutagens up to 100-fold lower than with other methods. In measuring mutation to tryptophan independence in *Escherichia coli* strain WR2 it was found that methyl methanesulfonate (0.5 μg/ml), mitomycin C (0.0015 μg/ml), dichlorvos (5 μg/ml), and K_2CrO_4 (0.5 μg/ml) were all positively mutagenic in the test, whereas $NiCl_2$ was negative. It appeared that chronic exposure to low levels of mutagens by this method induced more mutations than might be predicted by extrapolation from short exposure experiments at higher doses. This procedure is useful for any system involving mutation to prototrophy from a non-leaky auxotrophic requirement. It should prove of value in detecting and studying effects of low doses and chronic exposures.

76-2250. Schuepbach, M.; Hummler, H. (Dep. Exp. Med., F. Hoffmann-La Roche and Co., Basel, Switzerland). **Evaluation of the mutagenicity of ethylenethiourea, using bacterial and mammalian test systems.** *Mutat. Res.* 38(2): 122; 1976.

Ethylenethiourea (ETU), a metabolite and degradation product of ethylenebisdithiocarbamate fungicides, has been demonstrated to be carcinogenic, teratogenic, and a weak mutagen. Therefore it was used to provide information concerning the response of some common mutagenicity screening procedures to a possibly weak mutagen. In vitro reversion studies on different histidine-requiring *Salmonella typhimurium* strains were performed in a semi-quantitative plate test. A rise in the number of revertant stains was found with only the repair-deficient strain TA 1503. No frameshift responses were recorded. Thus a large fraction of the base-pair substitution type mutations were eliminated by excision repair. In the host-mediated assay a weak increase of the reversion frequency was noted only at 6000 ng/kg body weight using a single strain as indicator organism. Results in the micronucleus test on Swiss albino mice were negative. Only a slightly reduced fertility rate was observed in the highest dose group (3500 mg/kg) in the dominant lethal test on male mice. (From author abstract)

76-2251. Moutschen-Dahman, J.; Degraeve, N.; Houbrechts, N.; Colizzi, A. (Univ. Liege, Liege, Belgium). **Cytotoxicity of carbaryl alone and combined with nitrites.** *Mutat. Res.* 38(2): 122-123; 1976.

Even at low concentration, carbaryl induced a strong C-mitotic effect in *Nigella damascena* root tip cells; this effect disappeared a few hours after treatment. No chromosomal aberrations were detected in plant cells. When mice were given 0.2 ml per animal for seven days no C-mitotic effect was detected in bone marrow cells, nor was a chromosome breakage effect recorded. No effects were detected on mouse spermatogenesis, aside from some questionable sperm anomalies. If carbaryl is mixed with sodium nitrite immediately before administration to the animals no effects can be detected at the chromosome level but there are some mispairings in spermatogenesis. Similar conclusions were reached from experiments in which carbaryl was preincubated with sodium nitrite for one day before intubation to increase the degree of nitrosocarbaryl formation. (From author abstract)

76-2252. Sorsa, M.; Gripenberg, U. (Dep. Genet., Univ. Helsinki, Helsinki, Finland). **Organization of a mutagenicity test system combining instructive purposes: testing for mutagenic effects of the herbicide "amitrole."** *Mutat. Res.* 38(2): 132; 1976.

A simple mutagenicity screening program was developed involving three test organisms in five types of tests. Amitrole was selected for testing because of its wide use in Finland and its carcinogenic potentials. Amitrole did not increase the mutatation frequencies in any of the genetic or cytogenetic tests used or in any of the test organisms studied (*E. coli* and *Salmonella typhimurium*, *Drosophila melanogaster* in two types of genetic tests, and human lymphocyte culture cells). Negative results were obtained in the sex-linked recessive lethals tests in male Drosophila, and sex chromosome non-disjunctions were seen in females at a concentration of 0.001% in the medium. There was no significant increase in the occurrence of aneuploidy or structural changes found in the chromosomes of human lymphocyte cells even at subtoxic concentrations of amitrole (0.2% w/v.) (From author abstract)

76-2253. Seiler, J. P. (Swiss Fed. Res. Stn., Waedenswil, Switzerland). **Interactive mutagenicity of environmental chemicals.** *Mutat. Res.* 38(2): 135; 1976.

Many chemical mutagens are known, but few compounds have been tested for synergistic or antagonistic effects. The latter effect has been shown with fluoride in Drosophila and cell cultures. "Mutagenic synthesis" has been demonstrated to occur when nitrogenous substances and sodium nitrite were incubated at low pH, thus leading to the formation of *N*-nitroso-derivatives. By feeding mice with a mixture of ethylenethiourea and nitrite the incidence of micronucleated erythrocytes in the bone marrow could be greatly enhanced. The search for other pesticide-derived substances which would react in an analogous manner with nitrite revealed that most of the pesticides considered did produce mutations in bacteria but not in mammals. Another compound of possible significance is the insecticidal synergist piperonyl butoxide, which acts as an inhibitor of microsomal oxidases. Preliminary bacterial assays with this compound as well as with ascorbic acid, which has been proposed by Pauling for use in the "orthomolecular treatment" of cancer, will also be reported. (Author abstract by permission)

76-2254. Carere, A.; Cardamone, G.; Ortali, V.; Bruzzone, M. L.; Di Giuseppe, G. (Ist. Super. Sanit., Rome, Italy). **Mutational studies with some pesticides in *Streptomyces coelicolor* and *Salmonella typhimurium*.** *Mutat. Res.* 38(2): 136; 1976.

The purpose of this investigation was to study the mutagenicity of different classes of pesticides as pure compounds or commercial compounds using various genetic systems and the "plate test" technique. One system is based on the induction of resistants to low levels of streptomycin in the filamentous bacterium *S. coelicolor*; another system is based on the induction of revertants in a set of four histidine requiring mutants (TA 1535, TA 1536, TA 1537, and TA 1538) of *S. typhimurium*. The pesticides so far tested are the following: aminotriazole (amitrole), benomyl, captan, dinobuton, ioxynil, mecoprop, neburon, picloram, and tordon. Four pesticides (aminotriazole, dichlorvos, picloram, and tordon) induce streptomycin resistants in Streptomyces; up to now in Salmonella, doubtful results have been obtained with captan and benomyl while the other pesticides turned out to be non-mutagenic. (Author abstract by permission, abridged)

76-2255. Kappas, A. (Biol. Dep., Nuclear Research Centre "Democritus", Athens, Greece). **On the recom-**

binagenic action of certain fungicides in *Aspergillus* diploids. *Mutat. Res.* 38(2): 135-136; 1976.

A number of fungicides were found to interfere with the segregation of chromosomes at mitosis in *Aspergillus nidulans* diploids. This was shown by the increase of somatic segregation occurring in strains heterozygous for spore color mutations. Most of these fungicides induced mainly haploidization but they also increased the diploid segregants. In an attempt to understand the mechanism(s) responsible for the increase of mitotic segregation a prototrophic diploid strain was used heterozygous for several markers appropriate for the detection of new recombinations. Non-disjunction, mitotic crossing-over, and breakage-deletion were considered as possible mechanisms for the increased recombination by compounds belonging to two different groups of fungicides, benzimidazoles and aromatic hydrocarbons. It was indicated that depending on the fungicidal compound, different mechanisms might be responsible for the increase of recombination. (Author abstract by permission)

K76-2256. Egert, G.; Greim, H. (Dep. Toxicol., Univ. Tuebingen, Tuebingen, Germany). **Formation of dimethylnitrosamine from chloroxuron, cycluron, dimefox and thiram in the presence of nitrite.** *Mutat. Res.* 38(2): 136-137; 1976.

Tertiary amines can be nitrosated in the presence of nitrite under conditions which resemble those found in the stomach. Formation of such carcinogenic nitrosamines in the stomach may present a hazard to man since both amines and nitrite are known to be present in foods. Futhermore pesticide residues are also found in foodstuffs, including the tertiary amines chloroxuron, cycluron, dimefox, and thiram. Nitrosation of these pesticides was investigated in the presence of 6 *N* HCl or acetic acid at pH 1 and excess sodium nitrite for 4 hr at 37°C. The reaction products were extracted with CCl_4 and were analyzed both qualitatively and quantitatively by infrared spectrophotometry, nuclear resonance spectrometry, GC-mass spectrometry, and light spectrophotometry. All the pesticides investigated were found to form dimethylnitrosamine in the following yields: cycluron 7%, chloroxuron 8%, dimefox 28%, and thiram 16%. As expected, because of the well known mutagenicity of dimethylnitrosamine, extracts from the incubations of the different pesticides induced mutations in the *E. coli* K-12 strain in the presence of a metabolizing test system containing microsomes and a NADPH generating system. (Author abstract by permission)

76-2257. Aulicino, F.; Bignami, M.; Carere, A.; Conti, G.; Morpurgo, G.; Velcich, A. (Ist. Super. Sanit., Rome, Italy). **Mutational studies with some pesticides in *Aspergillus nidulans*.** *Mutat. Res.* 38(2): 138; 1976.

Induction of mutation and somatic recombination by the following pesticides has been studied utilizing *Aspergillus nidulans* as test organism: aminotriazole (amitrole), captan, dalapon, dichlorvos, dinobuton, dodine, ioxynil, mecoprop, neburon, picloram, and tordon (picloram as the triisopropanolamine salt). Point mutation has been studied as induction of 8-azaguanine resistants utilizing a spot test at three different pHs. Only captan turned out to be mutagenic. Induction of non-disjunction and mitotic crossing-over were studied with a diploid strain of the mold by: a) utilizing a spot test previously described at three different pHs, b) plating conidia of the mold on a medium supplemented with the maximum nonlethal dose of the pesticide and screening for the appearance of recombinant sectors in the colonies. Captan, aminotriazole, and dichlorvos induce somatic recombination. Especially noteworthy is the action of dichlorvos in inducing crossing-over. (Author abstract by permission, abridged)

76-2258. Bignami, M.; Conti, L.; Morpurgo, G.; Velcich, A. (Ist. Super. Sanit., Rome, Italy). **Comparative analysis of different test systems for somatic recombination with *Aspergillus nidulans*.** *Mutat. Res.* 38(2): 138-139; 1976.

Experiments are reported to test the validity of a previously described "spot test" to measure the induced somatic recombination (i.e. mitotic crossing-over and non-disjunction) with diploid strains of *Aspergillus nidulans*. The following results have been obtained: sulfanilamide, 5-fluorouracil, and vapona (dichlorvos) induce mitotic crossing-over both with the spot test and with non-selective tests; esidrex induces a very high frequency of non-disjunction using both the spot test and a non-selective technique. Enterovioform (Iodochloroxyquinoline) on the contrary induces non-disjunction with the spot test, while only doubtful results were so far obtained with the non-selective technique. (Author abstract by permision)

76-2259. Shirasu, Y.; Moriya, M.; Kato, K.; Furuhashi, A.; Kada, T. (Toxicol. Div., Inst. Environ. Toxicol., Kodaira-shi, Tokyo 187, Japan). **Mutagenicity screening of pesticides in the microbial system.** *Mutat. Res.* 40(1): 19-30; 1976. (24 references)

A study was made of the mutation induction capacity of 166 pesticides in the microbial system. Compounds studied included 57 fungicides, 63 herbicides, and 46 insecticides. Screening methods used included the rec-assay procedure, a sensitivity test utilizing H17 Rec+ and M45 Rec- strains of *Bacillus subtilis*, as well as reversion assays on plates utilizing auxotrophic strains of *Escherichia coli* (WP2) and *Salmonella typhimurium* (Ames series). Chemicals which induced reversions were noted only among those showing positive effects in the rec-assay and not among negative samples. Captafol, captan, dexon, and NBT, which had previously been shown to be mutagenic, were so demonstrated in these tests. In addition, dichlorvos, folpet, 2-hydrazinoethanol (HEH), 5-nitro-1-naphthonitrile (NNN), and vamidothion were found to be mutagens in these systems.

76-2260. Mathew, C.; Al-Doori, Z. (Biol. Res. Cent., Baghdad, Iraq). **The mutagenic effect of the mercury fungicide ceresan M in *Drosophila melanogaster*.** *Mutat. Res.* 40(1): 31-36; 1976. (13 references)

Mutagenic tests were conducted in Drosophila with the mercury fungicide, ethyl mercury-*p*-toluene sulfonamide, used to treat wheat seeds that were supplied to Iraqi farmers. The chemical was mixed with the food medium on which the Drosophila larvae were allowed to feed, in concentrations of of 30 and 40 ppm/100 cm^3 of food. The frequency of lethals after the 30-mg treatment was not markedly different from that in controls. But, after the 40-mg treatment, the frequency of lethals increased more than 5-fold. These results indicate that Ceresan M is clearly mutagenic in Drosophila with regard to the induction of sex-linked recessive lethals. The duration of the developmental period of Drosophila was also affected by the 40-mg treatment. It took at least 16 days for the adult flies to emerge from the treated medium, as against less than 12 days in controls, at 25°C. It thus appears that alkyl mercury compounds have definite effects on the genetic material.

76-2261. Dean, B. J.; Blair, D. (Shell Res. Ltd., Tunstall Lab., Sittingbourne Res. Cent., Sittingbourne, Kent, ME9 8AG England). **Dominant lethal assay in female mice after oral dosing with dichlorvos or exposure to atmospheres containing dichlorvos.** *Mutat. Res.* 40(1): 67-72; 1976. (18 references)

This communication describes the assay of dominant lethal mutations in female Carworth Farm No. 1 strain mice after oral dosing or inhalation exposure to dichlorvos. Mice were given single oral doses of 25 and 50 mg dichlorvos/kg, or i.p. injections of 100 mg/kg methyl methanesulfonate (MMS). Those exposed by inhalation were placed in test atmospheres for 8 weeks containing nominal concentrations of 2 or 8 μg of dichlorvos/l air. All female mice survived oral dosing with dichlorvos, exposure to atmospheres containing dichlorvos, or i.p. dosing with MMS without any detectable clinical effects. There were no significant effects on the percentage of pregnant females, the number of fetal implants, or the number of early fetal deaths in dichlorvos treated mice. Mice dosed with 100 mg MMS/kg had significantly fewer implants when mated during the first 5 days after dosing. A MMS dose of 100 mg/kg did not affect the pregnancy rate of CF1 females, but induced a reduction in implants and an increase in early fetal deaths, thus demonstrating the positive response of this strain to the induction of dominant lethal mutations by methylating agents. The results of this study, using various experimental approaches, did not demonstrate any induction of dominant lethal mutations in mice of either sex after dosing with high concentrations of dichlorvos.

76-2262. Herbold, B.; Buselmaier, W. (Inst. Anthropol. Humangen., Univ. Heidelberg, Heidelberg, Germany). **Induction of point mutations by different chemical mechanisms in the liver microsomal assay.** *Mutat. Res.* 40: 73-84; 1976. (10 references)

A selection of chemical agents with different mechanisms of chemical mutability was tested with the liver microsomal assay by using different bacterial tester stains, namely: *Salmonella typhimurium* TA 1535, TA 1536, TA 1537, TA 1538, and G46. The tested agents had been selected according to the following criteria. They are all well-known mutagens and can be divided into alkylating agents, anti-metabolites, acridines and those that form radicals in the cell. The mutagens were: dimethylnitrosamine (DMN), diethylnitrosamine (DEN), methylnitro-nitrosoguanidine (MNNG), cyclophosphamide, captan, amethopterine, azathioprine, 6-mecaptopurine, trypaflavine, isoniazine, and hydrazine. All except amethopterine gave positive results, showing that this system is very sensitive. In a comparison of the different strains and mutagens a correlation was found between the diameter of the molecule and the permeability of the bacterial cell membrane. (Author abstract by permission)

76-2263. Mahr, U.; Miltenburger, H. G. (Inst. Zool., Tech. Hochsch. Darmstadt, Darmstadt, Germany). **The effect of insecticides on Chinese hamster cell cultures.** *Mutat. Res.* 40: 107-118; 1976. (51 references)

The effect of *p,p'*-isomers of DDT and its derivatives DDD (TDE), DDE, and DDA, was studied on Chinese hamster cells in culture. At different concentrations and various times of treatment the proliferation rate was inhibited most strongly by DDD and DDT, whereas DDE exhibited a markedly weaker influence. DDA was the least toxic compound of the four. The cytogenetic effects were also different. Again, DDA induced the least damage. Only enhanced gap rates but no chromosome breaks were observed. DDE was more active, and higher break rates occurred. DDD and DDT were by far the most damaging compounds, and they raised the gap and break rates markedly. However, no induction of configuration anomalies was found in any experiment. Chronic treatment of the cells for 3 months with DDT at 8 ppm did not alter the proliferation rate, the sensitivity to acute treatment with higher DDT concentrations or the chromosomal aberration rates. The results are discussed in relation to the relevance of differential pesticide effectively in organs of higher animals and man. (Author abstract by permission)

76-2264. Lessa, J. M. M.; Becak, W.; Rabello, M. N.; Periera, C. A. B.; Ungaro, M. T. (Cent. Ciencias Biol., Univ. Fed. Alagoas, Alagoas, Brazil). **Cytogenic study of DDT on human lymphocytes in vitro.** *Mutat. Res.* 40: 131-138; 1976. (12 references)

The cytogenetic effect of DDT on human blood cultures in vitro was investigated. Two types of experiment were carried out: one in which the DDT concentrations found in the culture media were similar to those

found in the plasma of individuals of the Brazilian population (0.06-0.20 μg/ml); in the second experiments, doses ranging from 1 to 15 μg/ml were used. No correlation was found between DDT doses and cells with chromosomal aberrations. The Poisson test of comparison between means showed that at certain DDT concentrations (0.20, 4.05, and 8.72 μg/ml) the proportion of cells with structural aberrations was significantly greater than in the controls. (Author abstract by permission)

76-2265. Atkinson, G. (Dep. Botany, Auckland Univ., Auckland, New Zealand). **Effects of diquat on the microbiological organisms in the soil of lakes.** *N. Z. Dep. Sci. Ind. Res. Inform. Ser.* 97: 529-538; 1973. (10 references)

A study was made of the effect of diquat on organisms involved in the nitrogen cycle of lakes. Nitrogen fixation was measured by the acetylene reduction technique. The study showed that light was not an essential factor in the inhibition of nitrogen fixation by diquat. Diquat did not inhibit protein breakdown nor the reduction of nitrate to nitrite. However, diquat did inhibit the formation of nitrate from ammonia by *Nitrosomonas* and the reduction of nitrate to ammonia by *Desulfovibrio*. Diquat was reduced by a member of the genus *Achromobacter*. Free diquat persisted for at least 2 hr, as evidenced by dialysis experiments on the sediment. From the results of this study it was concluded that the 24-fold decrease in sensitivity of acetylene reduction in sediment may be due in part to the formation of the metabolically less active reduced radical. This reaction may be carried out by highly reductive dehydrogenases.

76-2266. Keogh, R. G.; Whitehead, P. H. (Grasslands Div., DSIR, Palmerston North, New Zealand). **Observations on some effects of pasture spraying with benomyl and carbendazim on earthworm activity and litter removal from pasture.** *N. Z. J. Exp. Agric.* 3(1): 103-104; 1975. (5 references)

The effects on pasture ecosystems of pasture spraying with benomyl and carbendazim against saprophytic fungal development were investigated. Benomyl (560 g a.i./ha) and carbendazim (560 g a.i./ha) were sprayed on a ryegrass-white clover, sheep-grazed pasture on 14 March 1974. Fungicide-treated plots had considerably fewer earthworm casts and significantly more litter than non-treated plots. The smaller number of casts on the fungicide-treated plots may not have been due to any marked reduction in the earthworm population; they are presumably a reflection of differences in the feeding activity of the worms. It is suggested that the slower rate of litter disappearance from the fungicide-treated plot is caused, in part, by a depression in earthworm feeding activity. The widespread use of pesticides which reduce earthworm activity particularly at critical periods should be seriously questioned.

76-2267. Egert, G.; Greim, H. (Dep. Toxicol., Univ. Tuebingen, Tuebingen, Germany). **Formation of mutagenic nitroso-compounds from ephedrine and from the pesticides carbaryl, dodin, and prometryn in the presence of nitrite at pH 1.** *Naunyn-Schmiedeberg's Arch. Pharmacol.* 293(Suppl.): R66; 1976.

Elespuru and Lijinsky have shown that drugs and pesticides carrying secondary amino groups undergo nitrosation in the presence of nitrite at moderately acid pH. These conditions resemble those found in the human stomach, because nitrite as well as drugs and pesticide residues are present in human food, including carbaryl, dodine, and prometryne. Nitrosation of these pesticides as well as of ephedrine was investigated in the presence of HCl and acetic acid at pH 1 and excess sodium nitrite for 4 hours at 37°. The reaction products were extracted with CCl_4 and were analyzed qualitatively and quantitatively by infrared spectroscopy, nuclear resonance spectrometry, GC/mass spectrometry, and by spectrophotometry. All compounds investigated formed *N*-nitroso derivatives in the following yields: ephedrine 4%, carbaryl 67%, dodine 12%, and prometryne 14%. The N-nitroso derivatives per se are not mutagenic to *E. coli* K-12. However, significantly increased mutation rates were seen after metabolic activation by mouse liver microsomes. These data add to the observations that nitrosoable compounds are present in the human environment. They may form potential mutagens in the human stomach in the presence of nitrite. Compounds giving high yields of nitrosoderivatives may present a health hazard to man and should be abandoned. (Author abstract by permission)

76-2268. Biczowa, B.; Sikorska, M.; Bicz, W.; Pachecka, J.; Sulinski, A. (Lab. Drug. Metab., Inst. Biopharm., Acad. Med. Warsaw, Warsaw, Poland). **Poziom glikogenu, glikoliza, oddychanie oraz stezenia ATP, ADP, AMP w mozgu szczura w nastepstwie zatrucia fosforoorganicznym insektycydem dichlorofosem (DDVP). [Glycogen level, glycolysis, respiration, and ATP, ADP, and AMP contents in rat brain following organophosphate insecticide (dichlorvos) intoxication.]** *Neuropathol. Pol.* 13(2): 259-272; 1975. (22 references) (Polish)

Changes in brains of rats intoxicated per o.s. by DDVP (dichlorvos) were monitored. Some rats were treated by a single administration of DDVP in doses of 2%, 10%, or 50% LD50. Others were intoxicated with identical doses of the pesticide over a period of 14 days. Still another group was intoxicated over a 90 day period with a 2% LD50 dose given daily. Glycogen levels were not changed following the single doses. An increase in glycogen was noted after 14 and 90 days of pesticide administration. The increase in glycogen content correlated with the dichlorvos dose given. No changes in aerobic and anaerobic glycolysis of rat brain homogenates were noted in the experiments. Oxygen uptake by the brain in the presence of glucose or succinate did not change after single or 14-day periods of administration. Chronic poisoning resulted in stimulated oxygen consumption in the presence

of succinate. No significant changes occurred in ADP or AMP content following either single or chronic intoxication. A small rise in ATP level was noted after 90 days of administration. The changes noted in this study may indicate that DDVP disturbs carbohydrate metabolism in the nerve tissue.

76-2269. Sitkiewicz, D.; Zalewska, Z. (Lab. Drug Metab., Inst. Biopharm., Acad. Med., Warsaw, Poland). **Aktywnosc oksydazy cytochromowej i dehydrogenazy bursztynianowej w mozgu szczura po zatriciu fosforoorganicznymi insektycydami dichlorfosem i trichlorofonem. [The activity of cytochrome oxidase and succinate dehydrogenase in rat brain mitochondria following trichlorfon and dichlorvos intoxication.]** *Neuropatol. Pol.* 13(2): 273-281; 1975. (20 references) (Polish)

Cytochrome oxidase and succinate dehydrogenase activities were studied in rat brain mitochondria after trichlorfon and dichlorvos (DDVP) intoxication. Insecticides were given p.o. at 2, 10, and 50% LD50. Effects were noted after one-time single-dose, 14 day, and 90 day periods of intoxication. Trichlorfon given as a single dose did not change the cytochrome oxidase and succinate dehydrogenase activity. After repeated trichlorfon intoxication at a 2% LD50 rate, the cytochrome oxidase activity was increased over a 14 day period. DDVP at all doses inhibited cytochrome oxidase activity while the succinate dehydrogenase activity was not changed over the 14 day period. Dichlorvos and trichlorfon did not affect succinate dehydrogenase activity when given for 90 days at small doses. The results of this study suggest that DDVP is more toxic for rat brain mitochondrial enzymes than is trichlorfon.

76-2270. Sikorska, M.; Biczowa, B.; Bicz, W.; Skonieczna, M. (Lab. Drug Metab., Inst. Biopharm., Med. Acad., Warsaw, Poland). **Glycogen content, glycolytic and respiratory activity, and ATP, ADP and AMP concentrations in rat brain following trichlorfon (dipterex) intoxication.** *Neuropatol. Pol.* 13(3/4): 447-454; 1975. (14 references)

The glycogen level, glycolysis, respiration, and adenine nucleotides concentrations in rat brain were studied following intoxication with trichlorfon. White male rats were given trichlorfon at 2, 10, and 50% LD50 in single or repeated doses. Both single and repeated administrations caused a statistically significant increase in glycogen levels in the cerebral hemispheres, depending on the dose applied. After 90 days of administration at a 2% LD50 dose, the glycogen level was elevated 14% over controls. Glycolytic activity of homogenates was not affected by trichlorfon intoxication. Lactate production and glucose consumption under aerobic and anaerobic conditions were similarly unaffected. No significant changes in the intensity of oxygen utilization in the presence of either glucose or succinate were noted following single dose intoxication or 14 day intoxication periods. A slight increase in brain ATP levels was noted after single-dose intoxication with a 50% LD50 dose. An increased brain ATP level also followed the 90-day intoxication with a 2% LD50 dose. The results of this study suggest that organophosphorus insecticides, besides inhibiting acetylcholinesterase activity, may cause disturbances of energy and carbohydrate metabolism in the central nervous system.

76-2271. Pachecka, J.; Sulinski, A.; Ziolkowska, G. (Lab. Drug Metab., Inst. Biopharm., 02-097 Warsaw, Poland). **The activities of some esterases of the rat brain after intoxication by organophosphate insecticides, dichlorvos and trichlorfon.** *Neuropatol. Pol.* 13(3/4): 455-462; 1975.

Effects of intoxication by DDVP (dichlorvos) and dipterex (trichlorfon) were examined on alkaline phosphatase, acid phosphatase, and acetylcholinesterase activities in rat brain homogenates. Male albino Wistar rats were used. Single-dose administration of DDVP in a 50% LD50 dose led to a decrease of alkaline phosphatase by 45%, of acid phosphatase by 16%, and of acetylcholinesterase by 58%. A 2% LD50 dose had no effect on the enzyme activities. Repeated administration of DDVP did not affect the alkaline phosphatase activity in either dose or time interval used. The acid phosphatase activity underwent a 25% reduction after 14 days of either 10 or 50% LD50 doses. Acetylcholinesterase activity dropped after a 14-day trial at 50 and 10% LD50 by 60 and 32%, respectively. Single-dose administration of dipterex in 50 and 10% LD50 doses caused reduced acetylcholinesterase activity by 74 and 55%, respectively. A 2% LD50 dose had no effect. Administration of dipterex for 90 days decreased acetylcholinesterase activity by 20% at the 2% LD50 dose level, and that of alkaline phosphatase by 18%. The results of these experiments indicate that intoxication by DDVP or dipterex, besides inhibiting acetylcholinesterase activity, affects the activity of brain phosphatases. This must be considered in evaluating the toxic effects of organophosphate insecticides on the central nervous system.

76-2272. Sitkiewicz, D.; Zalewska, Z. (Lab. Drug Metab., Inst. Biopharm. Med. Acad., Warsaw, Poland). **Effect of organophosphate insecticides on some oxidoreductases in rat brain mitochondria.** *Neuropatol. Pol.* 13(3/4): 463-469; 1975. (9 references)

Cytochrome oxidase and succinate dehydrogenase activities were examined in rat brain mitochondria in different experimental models after intoxication with dipterex (trichlorfon) or DDVP (dichlorvos). In in vivo experiments the insecticides were given to male albino Wistar rats p.o. at 2, 10, and 50% LD50. Single dose intoxication with dipterex at 2, 10, or 50% LD50 doses caused no change in the activity of cytochrome oxidase in the rat brain mitochondrial fraction. At the 2% dose level for 14 days, an increase in enzyme activity was noted. Dipterex for 90 days in small doses changed the assayed enzyme activity. DDVP produced inhibition of cytochrome oxidase activity after both single and multiple administration. After a single

dose of DDVP at the 50% LD50 dose level, cytochrome oxidase activity was not changed. However, repeating this dose for 14 days decreased enzyme activity. Intoxication with either chemical did not change the succinate dehydrogenase activity of rat brain mitochondria. In vitro studies showed that dipterex at 100 μg/mg protein inhibited cytochrome oxidase activity only after 15 min preincubation of the solubilized mitochondria with the insecticide. DDVP at all concentrations stimulated cytochrome oxidase activity without preincubation. The decrease of cytochrome oxidase activity after DDVP intoxication is not due to the direct action of the insecticide, but to the influence of its metabolites. Dipterex did not penetrate into the mitochondria. DDVP, however, apparently did penetrate mitochondrial membranes.

76-2273. Slizewski, M. (Dep. Neurol. Morphol. Lab., Inst. Nervous and Sensory Organs Diseases, Med. Acad., Bialystok, Poland). **Influence of chronic administration of phenylmercuric acetate on the peripheral nerve system of rat.** *Neuropatol. Pol.* 13(3/4): 471-477; 1975. (22 references)

Electrophysiological and pathomorphological changes were investigated in the peripheral nervous system of the rat after chronic administration of phenylmercuric acetate. Male Wistar rats were used. All animals received 1 ml of 0.3% solution of phenylmercuric acetate (0.25 LD50) each day through a stomach tube connected to a syringe. The results of the study demonstrated that organic compounds of mercury such as phenylmercuric acetate impair the peripheral nerves; in particular they evoke atrophy of the myelin sheath as early as the fourteenth day after administration. A slowing down of motor nerve conduction velocity in the sciatic nerves was noted after seven days of experimentation, with marked paresis of the hind legs noted after 35 days. The slowing of the motor nerve conduction velocity and pathomorphological changes preceded by far the clinical symptoms of impairment of the peripheral nervous system.

76-2274. Tokoro, T.; Suzuki, K.; Nakano, H.; Otsuka, J.; Suzuki, H. (Dep. Ophthalmol., Tokyo Med. Dent. Univ., Sch. Med., Tokyo, Japan). [**Experimental studies of organic phosphorus pesticide on beagle dogs. Long-term observations on the refraction and intraocular pressure.**] *Nippon Ganka Gakkai Zasshi (J. Jpn. Ophthalmol. Soc.)* 77(9): 1237-1245; 1973. (29 references) (Japanese)

The effects of organophosphorus pesticides were studied on beagle dogs. Ethylthiometon was given (p.o.) to dogs at dose levels of 5, 10, or 15 mg/dog/day for 2 yr. Dogs were examined 3 or 4 times during the study. Development of myopia was noted 14 months after the start of the study. Rule astigmatism was noted 5 months after the start of the study and was brought about by the corneal astigmatism. However, the rule astigmatism noted 14 months into the study seemed to be explained by the degree of both corneal and lens astigmatism. There was a significant correlation between the acetylcholinesterase level and the progress of myopia as well as between acetylcholinesterase and axial elongation; the lower the acetylcholinesterase level, the higher the degree of myopia and the greater the axial length. Intraocular pressure after instillation of tropicamide was notably elevated at 14 months, but had a tendency to be reduced after 24 months. These findings were considered similar to those of children's eyes which were diagnosed as showing chronic intoxication with organophosphorus pesticides.

76-2275. Matsushita, T.; Arimatsu, N.; Tomio, T. (Sch. Public Health, Fac. Med., Kumamoto Univ., Kumamoto, Japan). [**Experimental studies on the onset of allergic dermatitis due to dithiocarbamate pesticides.**] *Nippon Kogyo Eisei Gakkai Nenkai Hokoku (Proc. Jpn. Ind. Hyg. Soc. Annu. Meet.)* 49: 40-41; 1976. (Japanese)

Three experiments were performed to study the relationship between allergic dermatitis and contact with metallic salts of ethylene bis-dithiocarbamate. Guinea pig epidermis was examined 24 hr after contact with diluted solutions of maneb, mancozeb, zineb, thiram, ferbam, ziram, ethylene thiourea, sodium dimethyl dithiocarbamate, and lime-sulfur mixture to determine their stimulation threshold values (erythema levels). After sensitization by one of the above chemicals, another compound was used to determine allergenicity. Cross sensitization between compounds was also determined. Threshold values were: 5% or more for the first 4 compounds; 10% or more for the 5th, 6th, and 8th compounds; greater than 10% for the 7th compound; and 20% or more for the last mixture. Conspicuous onset of allergic dermatitis was indicated with all dithiocarbamates, especially with metallic salts of ethylene bis-dithiocarbamic acid. Definite cross reactions were recognized between metallic salts of ethylene bis-dithiocarbamic acid and those of dimethyl dithiocarbamic acid.

76-2276. Yamamoto, H. (Takeda Chem. Ind. Co. Ltd., Osaka, Japan). [**Validamycin, an antibiotic active against *Rhizoctonia solani*.**] *Noyaku Kagaku (J. Pestic. Sci.)* 3(4): 185-200; 1976. (47 references) (Japanese)

The development, properties, chemistry, production method, analysis, and safety of Validamycin A are described. Toxicity data shows that no mice or rats died after a single oral dose of > 20 g/kg, after subcutaneous administration of 15 g/kg, or after intravenous or intraperitoneal administration of less than 10 g/kg. It caused no irritation to rabbit skin and eyes in aqueous solution; when mice and rats were fed daily diet containing 0.1, 1, or 10% Validamycin, no abnormalities were observed other than diarrhea, soft feces, and swelling of the cecum in animals of the 10% group. During and after administration of 96 doses of Validamycin at 50, 200, and 800 mg/kg/day to beagles within four months, no change was observed. After oral administration of uniformly radiolabeled Validamycin 23.6% of the radioactivity was excreted via

exhaled air, 5.6% in urine, and 53.1% in feces within 72 hr. Microbial and soil degradation, residues in crops and soil, and environmental effects of Validamycin A were also described.

76-2277. Bimber, D. L.; Boening, R. W.; Sharma, M. L. (Dep. Biol., State Univ. Coll., Freedonia, NY 14063). **Respiratory stress in yellow perch induced by subtoxic concentrations of diquat.** *Ohio J. Sci.* 76(2): 87-91; 1976. (5 references)

Respiratory pressure changes, caused by the expansion and contraction of the buccal and opercular cavities during the respiratory cycle of the fish, were monitored by buccal catheterization prior to and after exposure to diquat in aquaria maintained in environmental chambers. Two-year-old yellow perch (*Perca flavescens*) from Chautauqua Lake in western New York State were studied on exposure to 1 and 5 ppm diquat. The data from the study indicates that for each individual on exposure to diquat, there was a statistically significant increase in the cough frequency at both concentrations. The concentrations of diquat used in the study are similar to those used in the lake during weed control measures and are significantly lower than those concentrations which cause death.

76-2278. Ben-Aziz, A.; Meisner, J.; Aharonson, N.; Ascher, K. R. S. (Agric. Res. Org., Volcani Cent., Bet Dagan, Israel). **Excretion of unchanged organophosphorus compounds after their consumption by larvae of the Egyptian cotton leafworm, *Spodoptera littoralis* Boisd.** *Pestic. Biochem. Physiol.* 6(1): 58-64; 1976. (12 references)

The ingestion and excretion of sublethal doses of phosfolan, monocrotophos, parathion, and leptophos were studied in larvae of the Egyptian cotton leaf worm, *Spodoptera littoralis* Boisd. On topical application, the difference in the toxicity of the compounds was not very large. The order from most toxic to least toxic was: phosfolan = leptophos > monocrotophos > parathion. The sipping method gave the following order of toxicity from most to least toxic: phosfolan, monocrotophos, leptophos, and parathion. At the sublethal concentrations tested, the recovery of leptophos from the feces ranged from 88 to 100% of the ingested quantity and that of parathion from 59 to 67%. Recovery of phosfolan and monocrotophos in the feces ranged from low (9.8 to 22.7%) to very low (3.9 to 6.7%) rates, respectively. The feeding rate was not markedly influenced either by the type of insecticide used or by the age of its residue. The study showed that the recovery from the feces of insecticides ingested by *S. littoralis* can be studied with facility by feeding the insects with insecticide plus phagostimulant-treated Styropor lamellae. Two of the insecticides tested, leptophos and parathion, were excreted either totally or at very high rates, whereas two others, phosfolan and monocrotophos, were excreted at low rates. The two compounds with low water solubility demonstrated a high excretion rate of the pure compound. For compounds with a high water solubility, the opposite effect was noted. The compounds with low water solubility were also less toxic.

76-2279. Dedek, W.; Lohs, K.; Fischer, G. W.; Schmidt, R. (Forschungsst. Chem. Toxicol., Acad. Wiss. DDR, Leipzig, DDR). **Alkylation of guanine in mice in vivo by organophosphorous insecticides. I. Trichlorphone and butonate.** *Pestic. Biochem. Physiol.* 6(2): 101-110; 1976. (61 references)

An investigation was made of the alkylating properties of trichlorfon and butonate toward the guanine N-7 of mouse nucleic acids in vivo. Male mice of strain AB Jena/Halle were used. The total dose per animal was 160 mg/kg for trichlorfon and 200 or 400 mg/kg for butonate. The compounds were labeled with ^{14}C on the OCH_3-group. The excretion rate of 7-MeG from nucleic acids was very rapid; contrary to the excretion rate of 3.0 to 3.5 days following administration of strongly genotoxic agents, half life of 2.0 hr was measured in liver for butonate and < 24 hr in the whole body for trichlorfon. The relative amounts of [7-^{14}C]methylguanine excreted in the urine were determined and compared with data for dichlorvos, dimethyl sulfate, and methyl methanesulfonate from the literature. Following i.p. administration, the methylating capability toward N-7 of guanine in nucleic acids is given by the ratio of about 100:10:25 for DDVP, butonate, and trichlorfon, respectively.

76-2280. Karel, A. K.; Saxena, S. C. (Toxicol. Lab., Dep. Zool., Univ. Rajasthan, Jaipur, India). **Chronic chlordane toxicity: effect on blood biochemistry of *Meriones hurrianae* Jerdon, the Indian desert gerbil.** *Pestic. Biochem. Physiol.* 6(2): 111-114; 1976. (19 references)

Chronic toxic effects of repeated doses of chlordane on the serum proteins, blood glucose, and serum alkaline and acid phosphatase activities of *Meriones hurrianae* Jerdon are reported. Healthy adult male gerbils were given repeated doses of 2.5 mg/kg chlordane, administered by i.m. injection in the thigh every 3 days for 45 days. Animals were autopsied at intervals of 15, 30, and 45 days following treatment. The effects of chlordane included hyperproteinemia, hyperglycemia, and enhanced serum alkaline and acid phosphatase activity. It is suggested that the increased incorporation of amino acids into proteins in the liver and the consequent release of proteins in the blood is the reason for the increased serum protein level in chlordane-intoxicated gerbils.

76-2281. Clark, A. G.; Cropp, P. L.; Smith, J. N.; Speir, T. W.; Tan, B. J. (Dep. Biochem., Victoria Univ. Wellington, Wellington, New Zealand). **Photometric determination of methyl parathion GSH S-methyl transferase.** *Pestic. Biochem. Physiol.* 6(2): 126-131; 1976. (12 references)

This report describes two methods for the assay of the enzyme which transfers a methyl group from methyl parathion to glutathione. The spectrophotometric assay

described is rapid, taking about 10 min, and is convenient for small numbers of assays. When large numbers of analyses must be run, the colorimetric assay described is more convenient, although it takes several hours. In sensitivity the spectrophotometric assay has a slight advantage; the effective value of ϵ for the colorimetric assay, after allowing for necessary fractions taken, is about 6000 and, although in the spectrophotometric method ϵ at 310 nm is only about 3000, a ten-fold expansion of scale is possible on the instrument used in this assay. In the colorimetric assay, the relative uncertainty of the substantial blank color does not permit working in a more sensitive spectrophotometer range, and it is necessary to arrange the enzyme concentrations to get absorbance readings 0.2-0.3 above the blanks, which are about 0.1. For crude enzymes, the sensitivity of the spectrophotometric assay is about 5 times as good. The utility of both methods has been demonstrated by the measurement of some kinetic constants and the distribution of the enzyme among some insects and vertebrates.

76-2282. Craven, A. C. C.; Brooks, G. T.; Walker, C. H. (Dep. Physiol. Biochem., Reading Univ., Whiteknights, Reading, England). **The inhibition of HEOM epoxide hydrase in mammalian liver microsomes and insect pupal homogenates.** *Pestic. Biochem. Physiol.* 6(2): 132-141; 1976. (26 references)

The effects are reported of several inhibitors on the hydrase activity toward the cyclodiene epoxide HEOM substrate in tissue preparations from two mammalian species and two insect species. Rat and rabbit liver microsomes and pupal homogenates of the blowfly (*Calliphora erythrocephala*) and the yellow mealworm (*Tenebrio molitor*) were compared as sources of the enzyme. Only minor differences were found between the four enzyme preparations, when considering *I*50 values and percentage inhibition at standard concentration. The simple epoxide 1,1,1-trichloropropane- 2,3-epoxide and two glycidyl ethers *p*-nitrophenyl glycidyl ether and *p*-ethylphenyl glycidyl ether tended to have lower *I*50 values ($1.8 \times$ m*M* to $8.0 \times$ m*M*) than triphenyl phosphate and SKF 525A ($4.5 \times$ m*M* to 1.40 μ*M*). Triphenyl phosphate and SKF 525A were competitive inhibitors for both the rat and *Tenebrio* enzymes. The only clear difference between these two epoxide hydrase preparations was with respect to their inhibition by 1,1,1-trichloropropane- 2,3-epoxide, which was an uncompetitive inhibitor with the rat enzyme, but showed kinetics of mixed inhibition with the insect preparation.

76-2283. Hinderer, R. K.; Menzer, R. E. (Dep. Entomol., Univ. Maryland, College Park, MD 20742). **Comparative enzyme activities and cytochrome P-450 levels of some rat tissues with respect to their metabolism of several pesticides.** *Pestic. Biochem. Physiol.* 6(2): 148-160; 1976. (38 references)

A study was made to compare the A- and B-esterases, amidase, and glutathione *S*-aryl transferase activities and cytochrome P-450 levels in blood and in microsomal, supernatant, and post-mitochondrial fractions of homogenates from lung, liver, kidney, and testis of the rat. These enzyme activities were correlated with the metabolism of several representative pesticides subject to both esteratic and oxidative attack: two insecticides, carbaryl and phosphamidon, and a herbicide, chlorotoluron. Male albino Sprague-Dawley rats were used in the study. The in vitro metabolism of carbaryl, phosphamidon, and chlorotoluron by the various centrifugal fractions revealed many differences. Carbaryl metabolism was greater in the liver microsomal fractions than in any other preparation. 1-Naphthol was the major metabolite in all tissue fractions. Although very little metabolism of phosphamidon occurred in the rat, metabolism in the rat liver postmitochondrial fraction was slightly higher with respect to the production of metabolites than in the supernatant and microsomes combined. Chlorotoluron was not metabolized by any tissue fractions of the rat. At least a low level of activity toward some compounds was noted in all tissues, but this study confirmed that the liver was the most active metabolizing tissue as well as having the highest levels of enzymatic activity usually associated with pesticide metabolism.

76-2284. Hinderer, R. K.; Menzer, R. E. (Dep. Entomol., Univ. Maryland, College Park, MD 20742). **Enzyme activities and cytochrome P-450 levels of some Japanese quail tissues with respect to their metabolism of several pesticides.** *Pestic. Biochem. Physiol.* 6(2): 161-169; 1976. (8 references)

This study was designed to compare the A- and B-esterases, amidases, and glutathione *S*-aryl transferase activities and cytochrome P-450 levels in blood and in microsomal, supernatant, and postmitochondrial fractions of lung, liver, kidney, and testis homogenates from the Japanese quail. These enzyme activities were correlated with the metabolism of two insecticides, carbaryl and phosphamidon, and a herbicide, chlorotoluron. In vitro metabolism of carbaryl, phosphamidon, and chlorotoluron by the various centrifugal fractions revealed that the production of 1-naphthyl-*N*-hydroxymethylcarbamate and 1-naphthol, the major metabolites, was greatest in the postmitochondrial fraction of the liver. The major carbaryl metabolite in all other quail tissue fractions was 1-naphthol. Phosphamidon metabolism in postmitochondrial preparations of quail liver was higher than in the supernatant and microsomes. Chlorotoluron metabolism occurred only in the post-mitochondrial fractions of quail liver. The major products were the oxidative metabolites, *N*-(3-chloro-4- methylphenyl)- *N'*-methylurea and *N*-(2-chloro- 4-hydroxymethyl- phenyl)-*N'*-methylurea.

76-2285. Haque, R.; Deagen, J. (Dep. Agric. Chem., Environ. Health Sci. Cent., Oregon State Univ., Corvallis, OR 97331). **Binding of bis(*p*-chlorophenyl) acetic acid with**

bovine serum albumin. *Pestic. Biochem. Physiol.* 6(2): 170-174; 1976. (14 references)

A study is presented of the interaction of DDA (a metabolite of DDT) with the protein bovine serum albumin (BSA). The binding of DDA with BSA was monitored by noting changes in the relaxation times of DDA proton magnetic resonance peaks. The addition of BSA to an aqueous solution of DDA increased line widths in a manner suggesting rapid, reversible binding. Line broadening was greater for the ring protons than for the benzylic proton, indicating that the ring presents the stronger binding site. The equilibrium constant was approximated. The binding results are considered in terms of environmental and toxicological factors. It is suggested that protein binding should be considered in discussions of the mode of action of DDT, as DDA is one of the major metabolites of that insecticide.

76-2286. Abston, P. A.; Yarbrough, J. D. (Dep. Zool., Mississippi State Univ., Mississippi State, MS 39762). **The in vivo effect of mirex on soluble hepatic enzymes in the rat.** *Pestic. Biochem. Physiol.* 6(2): 192-199; 1976. (32 references)

Tissue enzymes were examined relative to a mirex dose-response in a study to investigate the site of cellular enzyme loss and to measure other parameters which might be related to mirex exposure. Adult male and female Sprague-Dawley rats were fed mirex at 10, 20, 30, 40, and 50 ppm in the diet for 4 weeks. Significant decreases were noted in liver levels of lactic dehydrogenase, malic dehydrogenase, sorbitol dehydrogenase, glutamic oxaloacetic transaminase, and glutamic pyruvic transaminase. Enzyme losses were cytoplasmic and decreased in magnitude with increased exposure time and dietary mirex concentrations. Serum sorbitol dehydrogenase increased during the first week of mirex exposure while hepatic sorbitol dehydrogenase levels decreased. Hepatic glutamic oxaloacetic transaminase levels were significantly decreased during the first week at all concentrations as were the other enzymes at the 40 and 50 ppm concentrations. The magnitude of enzyme decreases in female rat tissues was consistently lower than enzyme decreases in the male. Liver weights for male rats increased significantly at all dietary levels during week one. However, only animals receiving 40 and 50 ppm levels showed significant liver weight increases at the end of week 4. Females showed liver weight increases at the 30 and 40 ppm levels after 4 weeks. No significant differences in body weights were noted for any level of mirex. Mirex hepatic residues appeared equal for both sexes. No gross histological alterations were noted after treatment.

76-2287. Nelson, P. A.; Stewart, R. R.; Morelli, M. A.; Nakatsugawa, T. (Dep. Entomol., State Univ. New York, Coll. Environ. Sci. For., Syracuse, NY 13210). **Aldrin epoxidation in the earthworm, *Lumbricus terrestris* L.** *Pestic. Biochem. Physiol.* 6(3): 243-253; 1976. (23 references)

Earthworms (*Lumbricus terrestris* L.) were injected with 10-50 μg of 1.0 m*M* to 10.0 m*M* aldrin in a Ringer solution. The aldrin was rapidly metabolized to dieldrin, which was not metabolized further. The observed epoxidation was not due to symbiotic bacteria. The aldrin epoxidase was located primarily in organs along the digestive tract, with most of the activity in the microsomal fraction of the intestinal tissue. The epoxidase activity increased gradually with development of the individual. Although sesamex did not inhibit the activity, inhibition of the epoxidase by carbon monoxide suggested the involvement of cytochrome P-450. However, the carbon monoxide difference spectrum of the microsomes was dominated by a material which was spectroscopically identical to earthworm hemoglobin. Verification of cytochrome P-450 in the earthworm microsome would require successful removal of this hemoglobin.

76-2288. Rosival, L.; Vargova, M.; Szokolayova, J.; Cerey, K.; Hladka, A.; Batora, V.; Kovacicova, J.; Truchlik, S. (Res. Inst. Hygiene, Bratislava, Czechoslovakia). **Contribution to the toxic action of *S*-methyl fenitrothion.** *Pestic. Biochem. Physiol.* 6(3): 280-286; 1976. (13 references)

The toxicity of the *S*-methyl isomer of fenitrothion to adult male Wistar rats treated by gastric lavage was higher than that of both fenitrothion and technical fenitrothion. Repeated administration of the compound led to a rapid decrease in toxicity with decreasing doses. Excretion of *p*-nitro-*m*-cresol in the urine of orally treated rats was more rapid after a single dose of the isomer than after fenitrothion; greater amounts of *p*-nitro-*m*-cresol were also excreted after treatment with the isomer. Single oral doses of the isomer increased the pentobarbitone sleeping time in mice at both 24 and 48 hr, but produced no change after 4 days. Administration of the isomer and purified fenitrothion to mice for 1 week had no effect on the pentobarbitone sleeping time, nor did a single dose of fenitrothion alter the effectiveness of pentobarbitone. Dominant lethal tests in rats revealed a possible mutagenic effect from the *S*-methyl isomer. The anticholinesterase activity of the isomer in vitro was 2-3 times higher than that of fenitrothion.

76-2289. Le Patourel, G. N. J.; Wright, D. J. (Dep. Zool. Appl. Entomol., Imp. Coll., London, England). **Some factors affecting the susceptibility of two nematode species to phorate.** *Pestic. Biochem. Physiol.* 6(3): 296-305; 1976. (12 references)

Two free-living soil nematodes, *Aphelenchus avenae* and *Panagrellus redivivus*, were treated with antibiotic solutions and incubated with aqueous solutions of (^{14}C)phorate. Both species showed coiling and reduced activity on exposure to phorate, but *A. avenae* was considerably more sensitive to the pesticide than *P. redivivus*. There was no significant mortality in either species after 24 hr exposure to 25 ppm phorate. *A. avenae* absorbed pho-

rate from an aqueous solution at a higher rate than *P. redivivus*, a phenomenon which was partially due to the greater total surface area per unit weight of the former. In contrast, the rate of phorate metabolism was much greater in *P. redivivus* than in *A. avenae*, and the oxidized products of phorate accumulated to a greater extent in *A. avenae* than in *P. redivivus*. The oxidized products of phorate in *P. redivivus* were phorate sulfone and phorate sulfoxide, whereas those in *A. avenae* also included phoratoxon sulfoxide. Phorate induced inhibition of acetylcholinesterase was greater in *P. redivivus* than in *A. avenae*. The differences in phorate metabolism exhibited by the two species thus account for the greater sensitivity to the compound shown by *A. avenae*.

76-2290. Ishaaya, I.; Engel, J. L.; Casida, J. E. (Div. Entomol. Parasitol., Univ. California, Berkeley, CA 94720). **Dietary triorganotins affect lymphatic tissues and blood composition of mice.** *Pestic. Biochem. Physiol.* 6(3): 270-279; 1976. (31 references)

Ph_3SnCl, Ph_3SnAc (brestan), Cy_3SnOH (plictran), and $(Bu_3Sn)_2O$ (TBTO) markedly reduced the splenic weight and blood weight gain of young male Swiss-Webster mice maintained for 7 days on diets containing 260 μEq organotin/kg diet. In mature mice maintained for 4 days on diets containing 780 μEq organotin/kg, these compounds resulted in a loss in splenic weight and body weight, a reduction in blood lymphocytes and total leucocytes, and an increase in erythrocytes, hemoglobin level, and hematocrit value. Four other organotins, (vendex), $PhSnCl_3$, Ph_2SnCl_2, and Ph_4Sn, were much less toxic to mice based on these parameters. Poisoning by the dietary triorganotins was not related to cerebral edema or alterations in adrenal epinephrine level, liver nonprotein -SH groups, or the activity of the intestinal and stomach proteases or intestinal amylase and invertase. The most sensitive indices of triorganotin toxicity appear to be those associated with changes in the lymphatic tissues and blood composition.

76-2291. Bushway, R. J.; Hanks, A. R. (Dep. Biochem., Texas A&M Univ., College Station, TX 77843). **Pesticide inhibition of growth and macromolecular synthesis in the cellular slime mold *Dictyostelium discoideum*.** *Pestic. Biochem. Physiol.* 6(3): 254-260; 1976. (22 references)

The effects of dieldrin and captan on the growth and macromolecular synthesis of the vegetative cells of *Dictyostelium discoideum* strain Ax-2 were investigated. Dieldrin at a concentration of 5 μg/ml inhibited growth as well as the synthesis of RNA, DNA, and protein, while as little as 1 μg/ml of captan produced the same effects. After a 1-hr exposure to either pesticide, all macromolecular syntheses ceased. Within a period of 5-10 hr the amebas began to shrink, and eventually some lysis occurred. Lysis was most pronounced in cells incubated with captan. When the amebas were grown in the presence of 5 μg/ml of either pesticide, then washed and resuspended in fresh medium, the effects on growth were annulled. No growth inhibition was observed when 0.05 *M* cysteine was added prior to the addition of 5 μg/ml captan. Breakdown of RNA and protein into TCA-soluble units did not occur in the presence of either pesticide. Preliminary studies have shown that (2-^{14}C)uracil and (^{14}C)amino acids are taken up in their respective pools in the presence of captan or dieldrin.

76-2292. Mathan, K. K.; Murugan, S. (Dep. Soil Sci., Tamil Nadu Agric. Univ., Coimbatore, India). **Influence of solvirex in combination with nitrogenous fertilizers on nitrification in the soil.** *Pesticides* 9)6): 27-29; 1975. (8 references)

A study was made on the influence of the organophosphate pesticide, solvirex, on nitrogen mineralization in soil. Solvirex 10 G is a granular systemic insecticide. Soils were treated with solvirex at the rate of 10, 20, or 30 kg/acre, at 10 kg/acre plus urea, at 10 kg/acre plus ammonium sulfate, or at 10 kg/acre plus sodium nitrate. The fertilizers were applied so as to supply 75 kg N/acre. The process of nitrification increased with increasing doses of Solvirex, but the pesticide caused depression in nitrification soon after its application up to 30 days. Nitrate production did not reach the level found in untreated soils even after 90 days in the case of soils receiving the rate of 10 or 20 kg pesticide/acre. Soils receiving 30 kg Solvirex/acre regained their original levels of nitrification within 45 days and registered an increase in nitrate production. Although the application of nitrogenous fertilizers along with the pesticide improved nitrate production, the applied rate of 75 kg N/acre was sufficient to reach the level of untreated control soil when applied as urea.

76-2293. Schwarz, F. P. (Jena, DDR). **Toxische Wirkung von Lindan auf Thrombozyten und Leukozyten der Taube. [Toxic activity of lindane on dove thrombocytes and leukocytes.]** *Pharmazie* 30(12): 808-809; 1975. (8 references) (German)

The effect of orally administered lindane on the thrombocytes and leukocytes of doves was studied. The blood smears were taken 2-3 days after administration of a single dose of 20 mg. Reduction of the thrombocyte and leukocyte counts was observed. The percentage of the destroyed cells was 47% against 19.2% (normal value) before the poisoning; the difference was statistically significant. The rate of destruction of the thrombocytes and leukocytes was increased even before the manifestation of CNS symptoms. There was practically no difference between intact and destroyed cells in the nuclear DNA content. Similar findings were obtained in chickens.

76-2294. Joshi, O. P.; Sachdev, M. S.; Sahrawat, K. L.; Kohli, B. N. (Nucl. Res. Lab., Indian Agr. Res. Inst., New Delhi, India). **Effects of simazine and atrazine on the mineralization of fertilizer and manure nitrogen.** *Plant Soil* 44(2): 367-375; 1976. (6 references)

In this study simazine and atrazine were treated for their effects on the mineralization of urea and sludge manure nitrogen in laboratory experiments. Sandy loam soil was used for the incubation studies. Simazine and atrazine were applied at 4 levels: 0.5, 1.0, 1.5, and 2.0 kg active ingredient/ha, with nitrogen applied at the rate of 200 ppm. In general, the application of simazine as well as atrazine increased the total amount of ammoniacal + nitrate nitrogen produced as compared to the untreated soils. The increase in the dose of herbicides did not show any specific trend in the mineralization of nitrogen, and these chemicals even at 2.0 kg/ha levels did not affect adversely the total mineralization of nitrogen or nitrate production compared to controls. Mineralization of total nitrogen (ammoniacal and nitrate nitrogen) in the presence of simazine and atrazine from the different sources in descending order was: urea > sludge + urea > sludge > no nitrogen.

76-2295. Skutilova, J. (Med. Fac., J.E. Purkyne Univ., Brno, Czechoslovakia). **Organicke slouceniny cinu, jejich toxicita a pouziti. [Organotin compounds their toxicity and their use.]** *Prac. Lek.*27(7-8): 267-271; 1975. (16 references) (Czech)

The mono-, di-, and tetraorganotin compounds are only very slightly toxic. The exceptions are methyl and ethyl compounds, which are biologicaly active. The greatest toxicity towards warmblooded animals and also towards microorganisms is displayed by triethyltin compounds. Bactericidal and fungicidal properties of tin are dependent on the organically bound forms of tin and not on the fungicidal properties of the tin itself, as is the case with the compounds of silver, for example. The acute toxicity of tributyltin in white mice and rats is similar to the toxicity of chlorinated insecticides; however, its toxicity towards zooplankton and cold blooded animals is much greater. The most important organotin compound is bis-tributyltin oxide. This compound is dangerous to aquatic animals (the greatest residual amount allowed in the river waters in Czechoslovakia is 1 μg). Bis-tributyltin oxide is also dangerous to humans, as it is easly absorbed by the skin and the mucous membranes. In warm blooded animals it caused blocking of the transmission of energy within the cell. In conclusion the author reports the cases of two persons. In one of these hematemesis occurred after exposure to bis-tributyltin oxide, and in the second person etching of the oropharynx occurred after consuming it.

76-2296. Yunghans, W. N.; Elder, J. H.; Wintersteen, B.; Morre, D. J. (Dep. Bot. Plant Pathol., Purdue Univ., West Lafayette, IN 47907). **Inhibition by DDT of calcium accumulation in isolated cell fractions from rat muscle.** *Proc. Indiana Acad. Sci.* 83:113-119; 1974. (22 references)

The effects of DDT on the accumulation of calcium by fragments of sarcoplasmic reticulum from skeletal muscle were investigated. Membrane vesicles of the sarcoplasmic reticulum fraction showed only scattered accumulations of electron-dense materials when incubated in the absence of calcium. The accumulation of calcium by sarcoplasmic reticulum membranes required adenosine-5'- triphosphate as an energy source. Spectrophotometric analysis confirmed that DDT inhibited accumulation of calcium by sarcoplasmic reticulum vesicles. Inhibition was not pronounced during the first minute of the reaction. No inhibition was noted at concentrations below 0.1 μM. Inhibition occurred between 0.1 μM and 5 μM DDT, and reached maximum near 5 μM DDT. Increasing the DDT concentration above 5 μM did not increase the degree of inhibition. The inhibition is expressed as a lag in the initial uptake of calcium and occurs at DDT concentrations similar to those which inhibit other ion transport systems. These results suggest that DDT interfered with membrane associated calcium transport to cause ataxia in poisoned animals.

76-2297. Gromisz, Z.; Leski, R. (Dep. Apiol., Gorna Niwa, Poland). **Szkodliwosc dzialania na pszczoly par preparatow Lebaycid, Folithion i Owadofos w warunkach polowych. [Toxicity of vapors of lebaycid, folithion, and ovadophos to bees under field conditions.]** *Pszczelnicze Zesz. Nauk.* 17: 197-204; 1973. (8 references) (Polish)

In several series of experiments on the toxicity of lebaycid (fenthion), folithion (fenitrothion) and ovadophos (also fenitrothion) at 0.15% concentrations conducted in 1967, 1968, and 1972, cages with honeybees were placed at different heights (0.5, 1.5 and 3.0 meters above ground) among apple trees either during spraying with the insecticides (direct contact) or 30, 60, 120 or 240 minutes after spraying (effect of vapors). Exposure times varied from 20 to 60 min. except for bees exposed to direct contact, which were removed immediately after spraying. All were transferred to "recuperation cages" and observed for 60 hours. Direct contact with the insecticides caused almost immediate death. The effect of vapors lingering in the area after spraying was weaker but also caused high mortality. At 26°C lebaycid was more toxic than folithion, vapors of lebaycid (30 minutes after spraying) causing death of 46.7% of bees exposed for 30 min., while folithion caused death of 24.7%. The toxic effect was dependent on the duration of exposure: vapors of lebaycid caused death of 2.6% of the bees after 20 min, of 52% after 40 min, and of 97.6% after 60 minutes of exposure, while mortality after exposure to folithion was 4.4%, 17.4% and 50.2% respectively (24 hour observation). At 26°C and 30°C the minimum exposure time causing significant mortality was 30 minutes, while 60 minutes caused a more than two-fold increase in mortality. Toxicity was greater at 0.5 and 1.5 m above ground than at 3 m, indicating that bees flying above tree tops were less endangered after application of insecticides. Elevations of humidity and temperature (application after rain at 30°C) caused an increase in the toxicity of ovadophos, resulting in 100% mortality within 6 hours when exposed for 60 minutes one hour after spraying. It was also noted that bees surviving exposure to vapors of these insecticides were much less active than controls; this would also have a strongly negative effect on their productivity in the beehive and fields.

76-2298. Satoh, T.; DuBois, K. P. (Res. Inst. Chemobiodynamics, Chiba Univ., Chiba, Japan). **A liver arylamidase extremely sensitive to organophosphorus compounds.** *Proc. Symp. Drug Metab. Action* 5: 163-173; 1973. (18 references)

This paper reports the effects of organophosphorus compounds on arylamidase and its microassay in human liver biopsy samples. The following compounds were tested for in vitro effects on liver arylamidase from male and female Sprague-Dawley rats: parathion, paraoxon, DEF, EPN, methyl parathion, sumithion (fenitrothion), Folex, TOCP, and malathion. Inhibition was remarkable with parathion, paraoxon, DEF, and EPN, and was also noted in the presence of high concentrations of methyl parathion, sumithion, Folex, and TOCP. For in vivo studies the animals received a single ip injection of sublethal doses of the organophosphorus compounds. Arylamidase was more susceptible to the relatively less toxic compounds such as sumithion, malathion, and TOCP than to the more toxic ones such as parathion, paraoxon, and EPN. The existence of a sex difference in the cases of parathion, paraoxon, and EPN can be explained by the correlation with the drug metabolizing enzyme activity associated with detoxification of these insecticides. Antiamidase activity was associated with alkyl substitutions in the chemical structures of insecticides. Microassay of an arylamidase in needle biopsy samples of human liver is also discussed. (Symposium held in Shizuoka, Japan, Nov. 9-10, 1973.)

76-2299. Personne, G.; Uyttersprot, G. (State Univ. Ghent, Lab. Biol. Res. Environ. Pollut., Ghent, Belgium). **The influence of inorganic and organic pollutants on the rate of reproduction of a marine hypotrichous ciliate: *Euplotes vannus* Muller.** *Rev. Int. Oceanogr. Med.* 37-38: 125-151; 1975. (15 references)

The toxicity of various organic and inorganic compounds to marine pelagic and benthic organisms is being studied as part of a Belgian interdisciplinary research program. The effects of lead, copper, mercury, zinc, and cadmium ions, as well as DDT, DDE, hexachlorobenzene (HCB), heptachlor, hexachlorocyclohexane (BHC), and Aroclor 1254 on the rate of reproduction of a common marine hypotrichous ciliate (*Euplotes vannus* Muller) were investigated. In contrast to most of the metal ions, the pesticides at 10 ppm caused only a 3-10% inhibition compared with control values. With Aroclor 1254, zero inhibition was observed at 10 ppm. It appears that the various North Sea sediments, especially in the vicinity of dumping sites, have considerably exceeded the metal ion toxicity threshold of *Euplotes vannus*.

76-2300. Ariens, E. J.; Simonia, A. M. (Pharmacol. Inst., Univ. Niimegen, The Netherlands). **Diseno de farmacos. [Drug design.]** *Rev. Quim. Teor. Apl.* 32(332): 1051-1086; 1975. (183 references) (Spanish)

The article provides an elaborate overview of ways to avoid drug toxicity and improve drug efficacy through the appropriate design of pharmaceuticals. An appreciation of the phases through which a drug passes from the time it is administered until it has acted within the human body is essential for effective drug design. Attention is paid to possible adverse effects of drugs and their contaminants, including those induced in the form of chemical lesions. Emphasis is placed on molecular manipulation as a means to ensure the manufacture of safer and better drugs. The synthesis of biodegradable DDT analogs serves as an example of the use of molecular modification to improve environmental characteristics of chemicals.

76-2301. Masuda, T. (Div. Work Environ. Occup. Dis., Inst. Sci. Labour, Tokyo, Japan). **[Studies of protein metabolism in rats under various conditions by means of determination of urinary vacat-O/N ratio. Part 3. Vacat-O/N ratio in rats administered phenylmercury acetate, dehydroacetic acid, and aqueous extract of tobacco.]** *Rodo Kagaku (J. Sci. Labour)* 51(11): 647-660; 1975. (23 references) (Japanese)

Results of studies on the effect of oral administration of phenylmercury acetate (pmA), dehydroacetic acid (DHA), and aqueous extract of tobacco (AET) are presented. Rats were given PMA (0.4 mg/day for 15 weeks) along with either vitamin B1 (VB_1 HCl) (1.25 mg/day) or thiamine disulfide (0.83 mg/day). Some rats received only PMA. The increase in body weight after 15 weeks' PMA administration suggested an edema caused by disturbance of water metabolism, as reduced function and hypertrophy of the kidney and increased water content of the muscles were noted. Most of the PMA accumulated in the kidney and only a small amount in the liver. However, the liver presented compensatory hypertrophy. VB_1 or thiamine disulfide attenuated the adverse effect of PMA. The vacat-O/N ratio was raised by PMA administration. Thiamine disulfide attenuated its rise, but VB_1HCl was ineffectual in this respect. In other experiments rats received DHA (20 mg/day) or AET (corresponding to 1 mg nicotine/day). Reduction of growth, decreased activity of liver enzymes, and compensatory hypertrophy of the liver were noted. DHA treated rats showed declines in tubular function and atrophy of the kidney. The vacat-O/N ratio was raised by DHA, especially in the early period of administration, suggesting that the biological burden was heaviest in this period. It is concluded from these studies that the vacat-O/N ratio is significantly raised by exposure to poisonous gases or by administration of poisonous substances, probably due to decreases in the protein metabolism resulting from excess burden to the liver function.

76-2302. Matsueda, S.; Nagaki, M.; Tsukada, N.; Sato, M.; Ishida, S.; Sasaki, K. (Dep. Gen. Educ. Sci., Hirosaki Univ., Hirosaki, 036, Japan). **[Therapeutic effect of chlorella extract (crude drug) and enzyme-histochemical findings on human blood cell cholinesterase inhibited by agricultural drugs.]** *Sci. Rep. Hirosaki Univ.* 20(1): 14-22; 1974. (17 references)

Chlorella extract may be used as a crude drug in cases of poisoning due to agricultural chemicals. In the course of experiments it was determined that the decrease in activity of human cholinesterase due to agricultural drugs was restored in vitro by chlorella extract. The use of this crude drug is not accompanied by apparent adverse effects. In these experiments the red blood cells inhibited by agricultural drugs were studied and those reactivated could be observed microsopically (x 1500). Blood samples from healthy volunteers were mixed with parathion, the desired antidote added, and the cholinesterase contents determined. Reactivation was not apparent at high parathion levels due to renewed inhibition. However, in more dilute solution, both pralidoxime and chlorella extract were effective ChE reactivators. Chlorella extract also reversed the externalization of the erythrocyte membrane caused by ChE inhibition, which was previously thought to be irreversible.

76-2303. Epstein, S. S. (Med. Sch., Case Western Reserve Univ., Cleveland, OH). **The carcinogenicity of dieldrin, part II.** *Sci. Total Environ.* 4(3): 205-217; 1975. (17 references)

By any standard definition, any agent which is conclusively shown to increase the incidence or shorten the latency of tumor induction in man or animals is a carcinogen. There is no precedent or scientific basis for the alleged distinction between carcinogens and "augmenting agents," and there is no justification for demanding information on basic mechanisms of action before classifying a chemical as carcinogenic. The variability in the spontaneous incidence of liver tumors in mice provides no obstacle to the demonstration of dieldrin-induced carcinogenicity in the liver or any other organ. Although dieldrin has not been tested for carcinogenicity in infant animals, infant animals are generally more susceptible to chemical carcinogens than adults. The relatively high exposure of infant animals to dieldrin is thus cause for concern. Mice exposed to dieldrin for limited periods early in life develop liver cancer late in life, the tumor incidence being dependent on the period of early exposure. Any prolongation of current exposure in man may therefore increase the degree of carcinogenic hazard. Exposure to dieldrin is involuntary and there is no basis for the recognition of "safe" levels of carcinogen exposure in human populations. There are therefore cogent scientific grounds to demonstrate that continued exposure of human populations to dieldrin constitutes an imminent hazard.

76-2304. Crocker, J. F. S.; Ozere, R. L.; Safe, S. H.; Digout, S. C.; Rozee, K. R.; Hutzinger, O. (Dep. Pediatr., Dalhousie Univ., Halifax, Nova Scotia, Canada). **Lethal interaction of ubiquitous insecticide carriers with virus.** *Science* 192(4246): 1351-1353; 1976. (23 references)

A study was made of the toxicity of specific solvent carriers used in insecticide formulations. Swiss white mice from an outbred strain (ICR) were used. Solutions of the chemicals were painted once daily on the mice from 24 hr after birth to day 11. The following solutions in corn oil base were applied: corn oil alone; pure DDT; commercial fenitrothion (which contains the emulsifiers and solvent); DDT plus the commercial fenitrothion; DDT plus 3.8% pure fenitrothion; and emulsifiers and solvent. After these exposures the mice were infected with a non-lethal dose of mouse encephalomyocarditis (EMC) virus. Most deaths occurred within 5 days of viral injection. Long-term survivors were observed until day 23, and were then killed. Mortality was much higher in animals exposed to the mixture of solvent and emulsifier and subsequently exposed to EMC virus than in those given corn oil alone or pure insecticides. Fenitrothion appeared to have a protective effect on this lethal interaction. DDT partially counteracted this protective effect. The small dose of virus required to produce death in these animals is below the LD50 for mice not primed by chemical toxin. It is concluded that the toxicity of these compounds could assume considerable significance for both inherent toxicity and enhancement of other agents of viral or toxic nature in exposed humans.

76-2305. Shirasu, Y. (Dep. Toxicol., Inst. Environ. Toxicol., Kodaira, Tokyo, Japan). **[Mutagens and teratogens.]** *Senten Ijo (Congenital Anomalies)* 15(4): 187-189; 1975. (Japanese)

Some pesticides, drugs, and other compounds including carcinogens were taken as examples for considering the relationship between mutagenesis and teratogenesis, which has not been so clearly elucidated as the relationship between mutagenesis and carcinogenesis. Described in detail was ethylenethiourea, the environmental decomposition product of many fungicidal ethylene bisdithiocarbamates such as zineb, maneb, etc., which comprises a pesticide residue in/on crops. It has been known that ethylenethiourea causes thyromas in rats and liver tumors in mice, and recently its teratogenicity has been demonstrated in rats. According to the mutagenicity results obtained using a microorganism system and dominant lethal assay, ethylene thiourea was only weakly active and only in *Salmonella typhimurium* TA1513. The above facts do not clearly indicate a relationship between mutagenicity and teratogenicity in ethylenethiourea. In the case of captan, the relationship was more complex, because it showed negative dominant lethality but it was strongly mutagenic in other systems. Its teratogenicity was positive in chick embryo and mouse, but was negative in rat, rabbit, and monkey.

76-2306. Teramoto, S.; Kaneda, M.; Shirasu, Y. (Dep. Toxicol., Inst. Environ. Toxicol., Kodaira, Tokyo, Japan). **[Teratogenicity of ethylenethiourea in rats. Part 1. Relationship of teratogenesis to dose rate and time of administration.]** *Senten Ijo (Congenital Anomalies)* 15(4): 234; 1975. (Japanese)

Single oral doses of ethylenethiourea (ETU) were given at respective dose rates of 100, and 200 mg/kg to

Wistar-Imanichi female rats on the sixth day to the fourteenth day of pregnancy. The relationship between the patterns of malformation and the time of administration was studied; the fetuses were removed and examined. No death of mother rats or fetuses was observed. Although no malformations were found after administration on days 6 to 8, various types of malformation were noticed after administration later than day 9. Major external malformations were meningocele, anal atresia (10th-13th day), microgenia, palatoschisis (11th-14th day), short tail, and oligodactylia (11th-13th day). Skeletal malformations were seen in skull and clavicle (11th-14th day), vertebra (9th-11th day), rib (9th-13th day), sternum (10th-14th day), and long tubular bones of the extremities (12th-13th day). Internal malformations included abnormalities of the cerebral ventricles (11th-14th day) and underdevelopment of the kidney (10th day). The higher the dose rate, the greater was the malformation frequency, with elongated critical period. The critical period was similar to that encountered in case of irradiation with X rays or antitumor agents.

76-2307. Teramoto, S.; Shingu, G.; Kaneda, M.; Saito, P.; Harada, T.; Kato, Y.; Shirasu, Y. (Dep. Toxicol., Inst. Environ. Toxicol., Kodaira, Tokyo, Japan). [**Teratogenicity of ethylenethiourea in rats. Part 2. Mode of teratogenic action of ethylenethiourea.**] *Senten Ijo (Congenital Anomalies)* 15(4): 234-235; 1975. (Japanese)

Ethylenethiourea (ETU) was administered orally at 100 mg/kg to Wistar-Imamichi female rats on the 12th day of pregnancy. The mode of teratogenic action of ETU was studied, paying particular attention to anomalies of the central nervous system such as enlargement of the cerebral ventricles. This phenomenon was observed in all the fetuses of treated mothers. By examining fetuses removed at various times it was found that although 6 hr after administration there was no difference from controls, necrosis of nerve cells along the neural cavity was seen at scattered points after 12 hr, and many necrotic nerve cells were seen all over the neural cavity. After 48 hr, active recovery began to be seen; however, cerebral development and differentiation were noticeably inhibited. Administration of radiolabeled ETU showed maximum radioactivity in the fetus after 2 hr. When ETU was injected into the amniotic cavity at 200 μg/10 μl and the fetus was removed on the 20th day of pregnancy and then examined, no anomaly was observed. When examined 48 hr after administration no necrosis of nerve cells was seen along the nerve cavity. On TLC plates of the extracts from fetuses of treated mothers, several unknown metabolites were found along with unchanged ETU.

76-2308. Lethbridge, G.; Burns, R. G. (Biol. Lab., Univ. Kent, Canterbury, Kent, England). **Inhibition of soil urease by organophosphorus insecticides.** *Soil Biol. Biochem.* 8(2): 99-102; 1976. (13 references)

The effect of three organophosphorus insecticides (malathion, fenitrothion, and phorate) on soil urease was examined. The inhibition of urea hydrolysis 60 days after the application of 1000 ppm insecticide to a sandy clay loam approached 40% (fenitrothion) and exceeded 50% in the case of malathion and phorate. Similar inhibitory effects were recorded using a silt loam soil in which 200 ppm insecticide produced inhibitions ranging from 14% (fenitrothion) to 23% (phorate) after 10 days. With lower insecticide concentrations (50 ppm) inhibition was more transient although still significant. All three insecticides in concentrations of 1000 ppm virtually prevented urea hydrolysis by jack bean urease. Ureolytic microorganisms, isolated from the soils under investigation, were inhibited by the organophosphates to a greater or lesser extent, but the development of tolerance was common. The application of insecticides to control soil-borne insect pests may be a factor in determining the efficiency of urea fertilizer mineralization.

76-2309. Jenkinson, D. S.; Powlson, D. S. (Rothamsted Exp. Stn., Harpenden, Herts., AL5 2JQ, England). **The effects of biocidal treatments on metabolism in soil. I. Fumigation with chloroform.** *Soil Biol. Biochem.* 8: 167-177; 1976. (38 references)

This paper contains a detailed examination of the effects of chloroform fumigation on a specific soil type. Chloroform fumigation immediately increased the ammonium and organic carbon extracted from the soil by 1*N* potassium sulfate. The chloroform-treated soil was then inoculated with fresh soil and incubated for ten days, after which time it consumed 2.8 times more oxygen, evolved 2.2 times more carbon dioxide, and mineralized 7.3 times more nitrogen than an unfumigated soil. The extractable organic carbon decreased by about 40%. A second fumigation immediately after the first caused no further increase in the flush, but if the soil was incubated between fumigations, some recovery was noted. A second fumigation 53 days after the first gave a flush only one-seventh the size of the first. Glucose added to the soil and decomposed before fumigation increased the size of the flush. After a 52-day incubation, 29% of the C originally added as ^{14}C labeled glucose was still present in the soil. Fumigation on the fifty-second day increased the evolution of ^{14}C-labeled carbon dioxide during the subsequent 10-day period 8 fold. Fumigation of a soil which had already been sterilized by 2.5 Mrad of gamma radiation increased the flush slightly, with the amount of oxygen consumed in 10 days increased from 123 to 137 mg/100 g soil. It is suggested that the flush of decomposition after chloroform fumigation is caused by the decomposition of killed organisms by the survivors and that organisms are more rapidly and completely attacked after chloroform exposure than after irradiation.

76-2310. Powlson, D. S.; Jenkinson, D. S. (Rothamsted Exp. Stn., Harpenden, Herts., AL5 2JQ, England). **The effect of biocidal treatments on metabolism in soil. II. Gamma irradiation, autoclaving, air-drying, and fumigation.** *Soil Biol. Biochem.* 8: 179-188; 1976. (29 references)

Respiration and mineralization of nitrogen were measured in a set of contrasting soils that had either been autoclaved, air-dried, fumigated, or exposed to gamma radiation. Soils used were a manured and an unmanured arable soil, an acid and a neutral woodland soil, an arable sandy soil, and an organic soil under grass. This paper compares the flushes caused by different biocidal treatments with that caused by chloroform fumigation and determines how soil factors such as pH or cropping history influence the size of the flush. The results of this study indicate that the fraction of the soil organic matter rendered decomposable by fumigation is associated with the entry of fresh plant material into the soil, rather than with the older, more stable parts of the soil organic matter. The Nigerian soils studied differed from the Rothamsted soils in that the fraction made decomposable by chloroform accounts for a larger percentage of the soil organic matter in the former. Thus, in the calcareous woodland soil from Rothamsted, 3.6% of the total soil N was mineralized in the 10 days following a chloroform treatment. The corresponding figure for the Nigerian forest soil was 4.9%. Another difference was in the ratio of N mineralized by fumigated soil/N mineralized by untreated soil. This was 12.2 for the Nigerian and 6.7 for the English woodland soil. However, this particular difference was caused partly by differences in the storage of the soil.

76-2311. Jenkinson, D. S.; Powlson, D. S.; Wedderburn, R. W. M. (Rothamsted Exp. Stn., Harpenden, Herts., AL5 2JQ, England). **The effects of biocidal treatments on metabolism in soil. III. The relationship between soil biovolume, measured by optical microscopy, and the flush of decomposition caused by fumigation.** *Soil Biol. Biochem.* 8: 189-202; 1976. (47 references)

This paper reports the measurement of the biovolume in a set of contrasting soils and compares the values obtained with those derived from the size of the flush of decomposition caused by chloroform fumigation. The distribution of the soil biovolume between hyphae and spherical organisms and between organisms of different sizes was also studied. The amount of biomass found from the flush of decomposition following fumigation was compared with the calculated biomass from direct microscopic measurement. For seven of the soils used there was close agreement between the results obtained by the two different methods of measuring biomass C. Both methods were used to study the effects of fumigation on the soil biomass. The decrease in biomass caused by fumigation was less when measured by direct microscopy than when measured from the size of the flush, suggesting that stainable cell walls of killed organisms can persist for a considerable time after fumigation. The biovolume in hyphae and in "spherical"organisms was roughly equal in all soils. There was a linear relation between the cumulative biovolume and the logarithm of organism volume for 'spherical' organisms over the volume range 0.05 μm^3 to 100 μm^3. Comparing equal logarithmic volume classes, a class of rare large organisms contains as much biovolume as a class of numerous small organisms.

76-2312. Jenkinson, D. S. (Rothamsted Exp. Stn., Harpenden, Herts., AL5 2JQ, England). **The effects of biocidal treatments on metabolism in soil. IV. The decomposition of fumigated organisms in soil.** *Soil Biol. Biochem.* 8: 203-208; 1976. (11 references)

To calculate the biomass from the size of the flush, a value must be assigned to k, the fraction of the killed biomass C that decomposes to carbon dioxide in the 10 days after fumigation. This paper reports an attempt to evaluate k by incorporating known amounts of different organisms into soil, fumigating, and then measuring the fraction of C mineralized from each organism when incubated under the standard conditions used to measure the size of the flush. Taking all the organisms studied together, 50.0% of the carbon originally present was mineralized in the 10 days after fumigation, giving a mean value of 0.5 for k. Experiments with earthworms showed that fumigation with chloroform did not appreciably increase the amounts of carbon and nitrogen mineralized from organisms that were already dead and the k was independent of the amount of organism added. It is concluded that the flush following fumigation can be used to give a rough measure of the amount of biomass in a soil.

76-2313. Jenkinson, D. S.; Powlson, D. S. (Rothamsted Exp. Stn., Harpenden, Herts. AL5 2JQ, England). **The effects of biocidal treatments of metabolism in soil. V. A method for measuring soil biomass.** *Soil Biol. Biochem.* 8: 209-213; 1976. (14 references)

In this paper a new method for the determination of biomass in soil is described. Soil is fumigated with chloroform vapor, the chloroform is removed, and the soil then incubated. The biomass is calculated from the difference between the amounts of carbon dioxide evolved during incubation by fumigated and unfumigated soil. The method was tested on nine soils from long-term field experiments. The amounts of biomass carbon/ha in the top 23 cm of the soil from plots on the Broadbalk continuous wheat experiment were 530 kg for an unmanured plot, 590 for a plot receiving inorganic fertilizers, and 1160 for a plot receiving farmyard manure. Soils fallowed for 1 year contained less biomass than soils carrying a crop. A calcareous woodland soil contained 1960 kg biomass carbon/ha and an unmanured soil under permanent grass contained 2020. The arable soils contained about 2% of their organic carbon in the biomass. Uncultivated soils carried a little more, about 3%. This technique has been used to measure the effects of pesticides on the soil biomass.

76-2314. Shpirt, M. B. (Kirgiz Sci. Res. Inst. Epidemiol. Microbiol. Hyg., Frunze, USSR). **K voprosu deystviya yadov na kletki cheloveka i teplokrovynkh zhivotnykh. [Effect on poisons on the cells of human beings and warm-blooded animals.]** *Sov. Zdravookhr. Kirg.* (6): 33-36; 1972. (Russian)

The toxicity of DDT, BHC, thiram, carbaryl (all at 100 mg/kg) and zineb (1,000 mg/kg) on human and rabbit

embryonal fibroblast cultures was studied. The various substances showed no significant differences in their action on human and rabbit fibroblasts. They exerted direct effects on the cells, causing cell degeneration, which was most pronounced under the conditions of nutrient deprivation. The cell degeneration indices in human fibroblasts were established to be 37.5% for DDT, 43.7% for BHC, 67.5% for thiram, 56.3% for carbaryl, and 56.3% for zineb. The LD50 of sevin for human embryonal fibroblasts was 191 mg/kg, and for rabbit fibroblasts 225 mg/kg. The cell cultures are much more sensitive to the pesticides than is the whole organism, which justifies their use in toxicological investigations and as a rapid test method.

76-2315. Kocmierska-Grodzka, D. (Dept. Pharmacol., Med. Sch., Bialystok, Poland). **Influence of trichlorfon and fractionated irradiation on hydroproteolytic activity of pancreas and intestinal tissues of rats.** *Strahlentherapie* 151(3): 278-284; 1976. (33 references)

A study was made of the effects of trichlorfon, given before fractionated irradiation, on the hydroproteolytic activity of some intracellular enzymes in pancreas and intestine of female Wistar rats. Trichlorfon was applied i.p. once a/day at a dosage of 10.0 or 30 mg/kg for 10 days before irradiation. Fractionated irradiation of the whole body was given for 150 R/day during 5 consecutive days for a total of 750 R. Experiments were conducted 24 hr after the last radiation exposure. Marked postirradiation enhancement of lipase activity was noted in the pancreas and duodenal part of the intestine. An increase in B-glucuronidase and acid phosphatase activities in nearly all parts of the examined intestinal tissues was also noted. The administration of trichlorfon evoked a modification of the hydroproteolytic activity in intestinal tissues of healthy and irradiated rats. Trichlorfon applied at the dose of 30 mg/kg exerted antilipolytic and anticatheptic effects in the pancreas and intestinal tissues of irradiated rats.

76-2116. Kimura, A.; Taniguchi, H. (Training Sch. Nurse Teachers, Tokushima Univ., Japan). **Influences of BHC, SMT, BHC+SMT, PCB, and FCPA at residual levels on effects of therapeutic agents.** *Tokushima J. Exp. Med.* 22: 11-19; 1975. (6 references)

The influence of PCBs on effects of therapeutic drugs was studied. Male dd-strain mice and male Wistar-strain rats were used. BHC, SMT (fenitrothion), FCPA (potassium-2-formyl- 1,4-chlorophenoxyacetate, a plant growth regulator),and PCB were used as the agricultural chemicals. The therapeutic agents studied included amobarbital sodium and hexobarbital sodium, which are hypnotics, and an analgesic agent, morphine HCl. The BHC+SMT group received 1.5 mg/kg/day BHC + mg/kg/day SMT. The BHC treated group received 1.5 mg/kg/day, and the SMT treated group received 5 mg/kg/day. Agricultural chemicals were injected i.p. The PCB treated group received 1.0 mg/kg/day and the FCPA group received 2.0 mg/kg/day. PCB was given orally, while FCPA was injected i.p. The influence of BHC+SMT on the effect of the drugs was highly significant as compared to that of the independently given chemicals. PCB shortened the hypnotic effect of hexobarbital sodium. The influence of PCB was not significant on the analgesic effect of morphine HCl, but it did provoke a tendency to augment this effect between the fourth and sixth weeks of the experiment. FCPA elicited a similar tendency to augment the effect of morphine HCl, but this was still insignificant.

76-2317. Lambert, G.; Brodeur, J. (Dep. Pharmacol., Fac. Med., Univ. Montreal, Montreal, Quebec, Canada). **Influence of starvation and hepatic microsomal enzyme induction on the mobilization of DDT residues in rats.** *Toxicol. Appl. Pharmacol.* 36(1): 111-120; 1976. (26 references)

Adult female Sprague-Dawley rats were treated with DDT (10 mg/kg po daily for 10 days) and were then treated with hepatic microsomal enzyme inducers and/or starved for various periods of time in order to study depletion of pesticide residues from the blood, brain, and abdominal fat tissue. The enzyme inducers used and the daily doses administered over a period of 14 days were: phenobarbital sodium 50 mg/kg, pregnene-16α- carbonitrile (PCN) 30 mg/kg, 9α-fluoro-11β, 17- dihydroxy-3-oxo-4-androstene-17α- propionic acid (CS-1) 40 mg/kg, and dexamethasone 4 mg/kg for 7 days and 2 mg/kg for 7 days. Starvation consisted in withholding solid food, but not water, for periods of 3 or 5 consecutive days, or 3 or 5 nonconsecutive days. At the end of the treatment period, the animals were sacrificed for determination of *p,p'*-DDT, *p,p'*-DDD, and *p,p'*-DDE. Phenobarbital and CS-1 reduced the concentration of the total residues in all three tissues examined, but phenobarbital was superior to the steroid. On the other hand, starvation for 5 consecutive days was more effective than any other period of starvation for reducing the concentration of the residues. Combination of a phenobarbital pretreatment of the residues. The degree of effectiveness of the combined treatment was better when the animals were starved early during the period of phenobarbital administration. These results provide good evidence that the combination of enzyme induction and starvation is a superior means of clearing contaminated tissues of DDT than either treatment alone. However, maximal efficacy is obtained only when both treatments are interrelated optimally with respect to the time and the duration of administration. (Author abstract by permission)

76-2318. Steen, J. A.; Hanneman, G. D.; Nelson, P. L.; Folk, E. D. (FAA Civil Aeromed. Inst., Oklahoma City, OK 73125). **Acute toxicity of mevinphos to gerbils.** *Toxicol. Appl. Pharmacol.* 36(1): 195-198; 1976. (9 references)

Intraperitoneal injections of mevinphos with doses ranging from 0.20 to 4.00 mg/kg were given to gerbils. Various somatic signs of poisoning, such as ataxia, piloerection, tremor, salivation, and convulsions, were noted. Somatic signs were observed in all cases; in all but two cases, effects were evident within 90 sec after injec-

tion. Animals were observed for at least 30 min following injection and, in lethal cases, time to death was recorded. LD50 values of approximately 0.45 mg/kg for males and 0.54 mg/kg for females were obtained. Females were more resistant to the chemicals. (Author abstract by permission)

76-2319. Polyakov, D. K.; Uzakov, U. Ya. (Lab. Arachnol. Acaricide Contr., All-Union Sci. Res. Inst. Vet. Hyg., USSR). **Nastavlenie po primeneniyu trikhlormetafosa-3 dlya bor'by s iksodovymi kleshchami na krupnom rogatom skote. [Instructions for the use of trichlorometaphos-3 in the control of ixodoid ticks in cattle.]** *Tr. Vses. Nauch. Issled. Inst. Vet. Sanit.* 43: 295-296; 1972. (Russian)

Instructions are presented concerning the application and handling of trichlorometaphos-3 in the control of ixodoid ticks in cattle. The animals are sprayed with 1% emulsion in water once every 7 days. The preparation maintains its acaricidal activity for seven days. Milk-cows must be excluded from the treatment. The necessary waiting period between the last treatment and slaughtering is 60 days. Protective clothes and a respirator with an air filter are required for workers handling trichlorometaphos-3. This preparation must not be stored and transported together with fodder, food, and water.

76-2320. Polyakov, D. K.; Frolov, B. A.; Khologov, I. Ya.; Uzakov, U. Ya.; Puchkova, E. A.; Speranskaya, V. N. (Lab. Arachnol. Acaricide Contr., All-Union Sci. Res. Inst. Vet. Hyg., USSR). **Vremennoe nastavlenie po primeneniyu sevina dlya bor'by s iksodovymi kleshchami i ektoparazitami ptits. [Preliminary instructions on the application of carbaryl for the control of ixodoid ticks and ectoparasites in fowl.]** *Tr. Vses. Nauch. Issled. Inst. Vet. Sanit.* 43: 297-300; 1972. (Russian)

Instructions are given concerning the application and handling of carbaryl in the control of ixodoid ticks in cattle and of ectoparasites in fowl. Cattle are sprayed with 0.85% suspension at an expenditure of 1-3 l/animal during the summer season, and dusted with 7.5% powder at a rate of 100-200 g/animal during the cold season. Poultry houses and poultry are sprayed with 0.1-0.25% suspension, or dusted. The necessary waiting period from the last treatment and slaughtering of cattle or poultry is 7 days.

76-2321. Boucard, M.; Orzalesi, H.; Serrano, J. J.; Peyre, P.; Tong, C. (Lab. Pharmacodyn., Pharm. Clin., Fac. Pharm., Montpellier, France). **Relations entre la structure chimique et la toxicite aigue de divers acides aryloxyacetiques substitues. [Relation between the chemical structure and the acute toxicity of various substituted aryloxyacetic acids.]** *Trav. Soc. Pharm. Montpellier* 34(2): 121-124; 1974. (French)

The toxicity of 21 compounds derived from phenoxyacetic acid was studied in 500 male Wistar rats. Most of the products had an acute toxicity of 400-500 mg/kg. In series A where only the influence of a substitution on the chain was considered, the toxicity decreased along with the molecular weight down to the aryloxy-2-methyl-2 propionic acids, then increased with the phenyl derivative. Any substitution on the nucleus was found to diminish toxicity in series C and E. In series B and D, the role of the position of the substitution on the nucleus was studied by maintaining on the most active constituent with *ortho* substitution and varying the position of the substitution in *meta* and *para*. While the *ortho* substitutions produced identical toxicities in both series, there was a complete inversion of LD50 between the *meta* and *para* derivatives. In series B, the *meta* derivative was less toxic than the *para*, and, in series D, it was the opposite.

76-2322. Neves, R. J. (Massachusetts Coop. Fish. Unit, Univ. Massachusetts, Amherst, MA 01002). **Zooplankton recolonization of a lake cove treated with rotenone.** *Trans. Am. Fish. Soc.* 104(2): 390-394; 1975. (12 references)

A study was undertaken in conjunction with a fish competition project to determine the time required for zooplankton to recolonize a lake cove treated with rotenone. South Branch Lake, located in Penobscot County, has a surface area of 824 hectares, with an average depth of less than 6 m. The cove studies has an area of 4.52 hectares and a mean depth of 1.6 m. Noxfish (5% rotenone) was applied from a boat to a concentration of about 0.6 ppm. Within 24 hr after application, net plankton volume dropped to 3% of pre-rotenone levels. Copepod and cladoceran populations were nearly exterminated after 2 days of toxic conditions. Rotifer populations also declined, although *Keratella* and *Conchilus* were not greatly affected. Entomostracans present in the toxic period, although in greatly reduced numbers, included *Eubosmina* sp., *Mesocyclops edax*, *Tropocyclops prasinus*, and *Copepod nauplii*. Limnetic species completely eradicated included the cladocerans: *Daphnia catawba*, *Daphnia dudia*, *Diaphanosoma leuchtenbergianum*, *Holopedium gibberum*, *Leptodora kindtii*, and *Polyphemus pediculus*; and the copopods *Epischura lacustria*, *Diaptomus minutus*, and *Cyclops vernalis*. The recolonization of the cover by limnetic zooplankters was rapid, with species abundances approaching those of open waters and of a control cove within one week.

76-2323. Zelinskiy, M. I. (Alma-Ata Veterinary Institute, Alma-Ata, USSR). **Toksichnost' khlorofosa i ego vliyanie na kachestvo myasa. [Toxicity of trichlorfon and its effect on the quality of meat.]** *Vestn. S'kh. Nauki Kaz.* 17(10): 66-68; 1974. (3 references) (Russian)

The effect of trichlorfon was investigated on the quality of the meat of rabbits. The animals were given 0.05 g of trichlorfon per kg of body through gastric probe in the form of 5% aqueous solution. The trichlorfon residue levels, determined 2-72 hours after slaughtering, were highest in the bones (12.4 mg), lower in the liver (7 mg), muscles (4.3 mg), brain (0.9 mg), and lowest in the kidney

(0.8 mg). When boiled in water, the meat released a mild, characteristic odor suggestive of trichlorfon. Reduced carbohydrate level and muscular enzyme activity, and increased pH were determined in the meat. Accumulation of amino acids and ammonia was observed.

76-2324. Volynchuk, I. M. (Kazakh Scientific Research Institute of Sylviculture, Kustanai Experimental Forestry Station, Kustanai, USSR). **Vliyanie gerbitsidov na biologicheskuyu aktivnost' pochvy. [Effect of herbicides on the biological activity of soil.]** *Vestn. S'kh. Nauki Kaz.* 17(4): 84-86; 1974. (Russian)

The biological activity was studied of chernozem treated with simazine (4, 6, 8 kg/ha), dalapon (15-20 kg/ha), and monuron (6-8 kg/ha). The accumulation of amino acids and the rate of destruction of linen in the soil were used as indicators of the biological activity. Both the cellulolytic bacteria and other microorganisms were depressed during the first month after pesticide application, which was followed by a normalization of their activity and even hyperactivity during the two vegetation periods covered by the experiment.

76-2325. Hammouda, M. H. M. (Alma-Ata Institute of Veterinary Sciences, Alma-Ata, USSR). **Deystvie gerbitsidov na svobodnozhivushchikh azotfiksiruyushchikh bakteriy. [Effect of herbicides on free nitrogen-fixing bacteria.]** *Vestn. S'kh. Nauki Kaz.* 17(4): 104-107; 1974. (5 references) (Russian)

The effect of 1-0.05% concentrations of ronit (cycloate), betanal (phenmedipham), chlorazone, sodium trichloroacetate (TCA), 2,4-D, and pyramin (pyrazon) on 63 strains of *Azotobacter* was studied in cell culture tests. The bacteria were most sensitive to sodium trichloroacetate, betanal, and 2,4-D, and less sensitive to pyramin and ronit. It was possible to develop about 30 strains which were resistant to the individual pesticides. The resistant bacteria were characterized by the ability to produce bacteriocins, and by the loss of their ability to form capsules and to synthesize vitamins B_1 and B_a.

76-2326. Hammouda, M. H. M. (Alma-Ata Institute of Veterinary Sciences, Alma-Ata, USSR). **Biokhimicheskaya aktivnost' svobodnozhivushchikh azotfiksiruyuschikh bakteriy, ustoychivykh k gerbitsidam. [Biochemical activity of free-living nitrogen-fixing bacteria resistant to herbicides.]** *Vestn. S'kh. Nauki Kaz.* 17(8): 109-112; 1974. (3 references) (Russian)

The biochemical activity was studied of *Azotobacter* strains resistant to pyramin (pyrazon), chlorazone, 2,4-D, ronit (cycloate), and betanal (phenmedipham). Compared with the original strains, the resistant strains had a reduced capacity to decompose carbohydrates, especially mannitol and sucrose. The growth rate of the resistant strains was highest in Ashby culture medium with sucrose, but their growth stopped more or less completely when sucrose was replaced by glucose. Alanine and arginine induced the synthesis of a yellow pigment, but histidine, methionine, threonine, lysine, leucine, and valine did not. The resistant strains grow best in the presence of those herbicides to which they are resistant, but at a lower rate than in the absence of herbicide.

76-2327. Kuul', A. K. (All-Union Sci. Res. Inst. Exp. Vet. Med., USSR). **Diagnostika otravleniya zhivotnykh ftalofosom. [Diagnosis of phthalophos poisoning in animals.]** *Veterinariya (Moscow)* 12: 103-104; 1975. (Russian)

The toxicity of phthalophos (phosmet) was studied in rabbits and chickens fed 50-600 mg/kg doses. The toxic effect of phthalophos manifested itself in functional disturbance of the sympathetic nervous system (miosis, bronchial spasm, sialorrhea, tremor, spasma, areflexia, diarrhea, asphyxia), dehydration, thickening of the blood, and extensive reduction of the acetylcholinesterase and Na,K-ATPase activities. The pathomorphological examination revealed hemodynamic, dystrophic and inflammatory-necrotic changes. The above symptoms as well as the determination of pthalophos in the gastric content and organs are pathognomonic.

76-2328. Tishkov, A. I. (All-Union Scientific Research Institute of Non-Infectious Diseases of Animals, USSR). **Kliniko-morfologicheskie izmeneniya i diagnostika otravleniya zhivotnykh preparatami rtuti. [Clinical-morphological changes and diagnosis of poisoning by mercurial preparations in animals.]** *Veterinariya (Moscow)* 1: 96-97; 1976. (Russian)

Four-month-old cocks were fed granosan (0.1 g/day) and/or agrosan (0.1 g/day) for 8-16 day in a study on the toxicity of mercurials. An initial hyperexcitability was followed by depression, asthenia, tremor, paresis, paralysis, blindness, anorexia, hypersalivation, emesis, diarrhea, and cyanosis. Stomatitis, pharyngitis, esophagitis, leukocytosis, thromobocytopenia, reduced serum protein level, and inflammatory, dystrophic, and necrotic changes in the liver, kidneys, and brain were found. Mercury was found in the liver, kidneys, myocardium, skeletal muscles, spleen, esophagus, trachea, lungs, brain, bones, gastrointestinal tract, pancreas, blood, skin, feathers, and testes. Mercury levels of 0.001-0.049 mg% were found in the internal organs of albino mice, guinea pigs and rabbits fed aerial parts of wheat and barley plants grown from seeds dressed with agrosan for 6 months.

76-2329. Konyukhov, A. F.; Poloz, D. D.; Kuul', A. A. (All-Union Scientific Research Institute of Experimental Veterinary Medicine, USSR). **Soderzhanie ftalofosa v organakh krolikov pri ostrom otravlenii. [Phthalophos levels in organs of rabbits following acute poisoning.]** *Veterinariya (Moscow)* 2: 92-94; 1976. (Russian)

Chinchilla rats were sacrificed 2 hours to 20 days after poisoning with 100 or 50 mg/kg oral doses of

phthalophos (phosmet) to investigate the distribution of the poison in different organs. General depression, reduction of muscle tones, miosis, sialorrhea, tremor, fasciculation of the dorsal muscles, spasm of the limbs, dyspnea, cyanosis, and coma were observed. The acetylcholinesterase activity dropped by 87%. Passive hyperemia of the blood vessels of the brain and parenchymatous organs was revealed on autopsy. Phthalophos and an unidentified metabolite with $Rf = 0.72$ were found in the gastric content and in the liver of animals killed 2 hours after poisoning. Phthalophos was no longer detectable in the gastric content of animals killed more than 4 days after poisoning, while residues appeared on the fourth day in the skeletal muscles and brain. Normalization of the acetylcholinesterase activity and disappearance of phthalophos residues from the organs was observed in animals killed on the 20th day.

76-2330. Polishchuk, L. R.; Matvienko, I. N. (Kiev Scientific Research Institute of Food Hygiene, Ukrainian SSR Ministry of Public Health, Kiev, USSR). **Raspredelenie ostatochynkh kolichestv DDT v organakh i tkanyakh ryb kievskogo vodokhranilishcha. [Distribution of DDT residues in fish organs and tissues in Kiev reservoirs.]** *Vop. Pitan.* 1: 82-83; 1976. (18 references) (Russian)

Fat, gonads, and liver of pike-perch, pike, perch, bream, and roach, caught in a reservoir near Kiev, USSR, during the winter and spring season, were analyzed for residues of DDT and its metabolites. DDT and its metabolites were found in most organs and tissues. The residue levels, increasing with age, were highest in the fat, and lower in gonads and livers, and they were higher in ichthyophagous species than in herbivora. Young specimens were free from residues or contained only traces of DDT and its metabolites. Except for pike-perch, all other species were free from residues in their muscles. The total DDT level (DDT+DDE+DDD) ranged from 0 to 5.75 mg/kg.

76-2331. Dragomirescu, A.; Raileanu, L.; Ababei, L. (Inst. Hyg. Pub. Health, Dep. Biochem., Inst. Med., Iasi, Romania). **The effect of carbetox on glycolysis and the activity of some enzymes in carbohydrate metabolism in the fish and rat liver.** *Water Res.* 9(2): 205-209; 1975. (18 references)

The effects of an indigenous malathion-containing organophosphorus pesticide, carbetox, were studied on the glycolytic capacity of the fish and rat liver and on some enzymes involved in the metabolism of carbohydrates. The pesticide, containing 37% malathion as active ingredient, was used in a 3:1000 solution, the dilution in which it is completely soluble and is used for spraying trees. Experiments were conducted on *Cyprinus carpio* L. fry and on albino rats. Carbetox decreased the capacity of the liver to form lactic acid significantly in both fish and rats. In vitro experiments showed similar conclusions. The activity of glucose-6-phosphate dehydrogenase was also reduced in the rat liver after intoxication. Lactate dehydrogenase exhibited a low activity after intoxication. In vitro experiments showed that carbetox strongly inhibited the activity of LDH in both fish and rat liver. It is noted that the degree of inhibition of carbetox for G-6-PDH, LDH, and G-6-Pase activity in the liver is dependent on the concentration.

76-2332. Carlson, W. C.; Lignowski, E. M.; Hopen, H. J. (Chemagro Div., Baychem Corp., Monticello, IL 61856). **Uptake, translocation, and adsorption of pronamide.** *Weed Sci.* 23(3): 148-154; 1975. (26 references)

Pronamide [3,5-dichloro- *N*-(1,1-dimethyl -2-propynyl)benzamide] was most phytotoxic to oat (*Avena sativa* L.) when placed in the seed zone and to quackgrass (*Agropyron repens* (L.) Beauv.), when placed in the rhizome zone. Inhibition of part of the buds by pronamide on detached quackgrass rhizome sections did not influence other buds. Foliar applications of pronamide were not phytotoxic to established oat or quackgrass. Foliar-applied ^{14}C-pronamide showed little uptake by quackgrass. Application to the roots of established plants showed rapid root uptake and movement to the foliage. ^{14}C-Pronamide was rapidly absorbed by oat seedlings for the first 0.5 hr and subsequently at a much slower rate. Over 60% of the pronamide taken up after 1 hr was exchangeable. Pronamide was adsorbed to the cell walls of treated roots, but little was associated with nuclear, mitochondrial, microsomal, or soluble protein fractions. Pronamide was adsorbed to varying degrees depending on soil type. Adsorption was more highly correlated with soil organic matter content than with cation exchange capacity, clay content, or pH. (Author abstract by permission)

76-2333. Golab, T.; Althaus, W. A. (Lilly Res. Lab., Greenfield, IN 46140). **Transformation of isopropalin in soil and plants.** *Weed Sci.* 23(3): 165-171; 1975. (19 references)

Field soil studies with ^{14}C-isopropalin (2,6-dinitro-*N*,*N*- dipropylcumidine) have demonstrated its biodegradability. None of the recognizable transformation products in soil accumulated beyond 4% of the amount of applied herbicide. After 15 months, approximately 25% of the applied isopropalin remained in soil, and less than 10% was observed after 28 months. Only negligible amounts of radioactivity were detected in crops grown in soil treated with the herbicide. Isopropalin and its recognizable transformation products were not detected in any of the plant tissues examined. Fractionation techniques indicated that the radioactivity was randomly distributed throughout the plant tissue. (Author abstract by permission)

76-2334. Mahoney, M. D.; Penner, D. (Dep. Crop Soil Sci., Michigan State Univ., East Lansing, MI 48824). **Bentazon translocation and metabolism in soybean and navy bean.** *Weed Sci.* 23(4): 265-271; 1975. (11 references)

Experiments were conducted to determine the movement of ^{14}C from ^{14}C-bentazon in soybean and navy

bean, examine differences in translocation of ^{14}C between unifoliate and first trifoliate leaves of navy bean, determine the influence of herbicide pretreatments on bentazon translocation and metabolism, and compare bentazon metabolism in soybean and navy bean. The movement of the ^{14}C-label was primarily acropetal in both species, with some basipetal movement. Metabolism was more rapid in the trifoliate soybean leaf. Prior treatment with several other herbicides did not serve to decrease metabolism of bentazon in the navy bean. The metabolites in the navy bean and soybean appeared to be similar. Four apparent ^{14}C-conjugates were isolated from soybean plants harvested at full maturity. It is suggested that the metabolism and translocation of bentazon appear to be major factors in the greater tolerance of navy bean in the trifoliate stage of growth. There is an indication that other factors contribute to the tolerance of soybean to foliar applications of bentazon.

76-2335. Mahoney, M. D.; Penner, D. (Stauffer Chem. Co., Mountain View, CA). **The basis for bentazon selectivity in navy bean, cocklebur, and black nightshade.** *Weed Sci.* 23(4): 272-276; 1975. (7 references)

The basis for the selectivity of bentazon between navy bean at two stages of growth and two weed species, cocklebur and black nightshade, was studied by comparing spray retention on the leaves, translocation, and metabolism. The plants were grown from seed and bentazon was applied at a rate of 2.24 kg/ha in 934 L/ha to cocklebur and black nightshade, having 4 to 6 leaves, and to navy bean having only unifoliate leaves or both unifoliate and trifoliate leaves. The trifoliate leaf of tolerant navy bean retained less bentazon from foliar applications than the unifoliate leaf, or the leaves from susceptible black nightshade seedlings. The ^{14}C from ^{14}C-bentazon applied to the foliage of black nightshade moved throughout the entire plant. In cocklebur, the labeled carbon moved throughout the treated leaf, whereas in the trifoliate leaf of navy bean very little acropetal movement occurred from the site of application. However, in the unifoliate leaf of navy bean, acropetal and basipetal movement of ^{14}C from the leaf was noted. The species differed in the 14C-metabolites formed from ^{14}C-bentazon 1 and 5 days after treatment. The rapid metabolism of bentazon in the trifoliate leaf of navy bean appears to be related to tolerance.

76-2336. Kern, A. D.; Meggitt, W. F.; Penner, D. (Dep. Crop Soil Sci., Michigan State Univ., East Lansing, MI 48824). **Uptake, movement, and metabolism of cyanazine in fall panicum, green foxtail, and corn.** *Weed Sci.* 23(4): 277-282; 1975. (17 references)

This study determined the contribution of foliar and root absorption, translocation, and metabolism to cyanazine toxicity and selectivity. The nature of the metabolism appeared different among the species studied. Corn harvested 5 days following treatment had more distinguishable metabolites than did fall panicum or green foxtail. Fall panicum contained a metabolite in the aqueous fraction which moved only slightly from the origin and could not be separated from it. Greater absorption and less metabolism accounted for the high concentration of parent cyanazine in fall panicum both 1 and 5 days after foliar treatment. Less absorption and a lower concentration of unaltered cyanazine in corn appeared to be the basis for selectivity to foliar treatments.

76-2337. Gingell, R. (Eppley Inst. Res. Cancer, Univ. Nebraska Med. Cent., Omaha, NB 68105). **Metabolism of ^{14}C-DDT in the mouse and hamster.** *Xenobiotica* 6(1): 15-20; 1976. (12 references)

This report presents a quantitative study of several urinary metabolites after a single exposure of CF-1 mice and Syrian golden hamsters to ^{14}C-DDT, and describes a new conjugate of DDA with serine. Six CF-1 mice and 3 Syrian hamsters were given ^{14}C-DDT (25 mg/kg, 1 and 5 μCi, respectively, in olive oil) by gavage and maintained in all-glass metabolism cages. DDT was metabolized in both mouse and hamster to DDA and conjugates of DDA. Apart from those conjugates with glucuronic acid, glycine, and alanine previously reported, a new conjugate with serine was now identified. DDT metabolism appears different in mice than in hamsters and some other species reported in that small but significant amounts of DDE were excreted in mouse urine, whereas none was noted in the hamster. The results of this study suggest that DDE may be a proximate hepatotumorigenic metabolite formed in mice fed DDT.

76-2338. Bolt, H. M.; Kassel, H. (Inst. Toxicol., Univ. Tubingen, D-74 Tubingen, Germany). **Effects of insecticide synergists on microsomal oxidation of estradiol and ethynylestradiol and on microsomal drug metabolism.** *Xenobiotica* 6(1): 33-38; 1976. (21 references)

This paper reports the interaction of some compounds, originally designed as insecticide synergists, with the microsomal metabolism of the estrogenic hormones estradiol and ethynylestradiol. The study showed that the oxidation of estradiol and ethynylestradiol at ring A and ring B by rat liver microsomes and NADPH-regenerating system in vitro was inhibited by the arylimidazole insecticide synergists: 3-bromophenyl- 4(5)-imidazole and 1-naphthyl- 4(5)-imidazole. However, no such inhibition was noted with the benzothiadiazole insecticide synergists: 6-nitro-1,2,3- benzothiadiazole and 5,6-dimethyl-1,2,3- benzothiadiazole. The *Ki* of the most potent inhibitor, 1-naphthyl-4(5)-imidazole, was 3 μM. 6-Nitro-1,2,3- benzothiadiazole (1.0 mM) did not inhibit hydroxylation of estrogens, but did inhibit oxidation of aniline and demethylation of ethylmorphine, *p*-nitroanisole, and aminopyrine by 30-70%. Only demethylation of *p*-nitroanisole and aminopyrine was inhibited by 5,6-dimethyl-1,2,3- benzothiadiazole. These results suggest that different hepatic microsomal mixed function oxidases may exist. The most potent inhibitor of hydroxylation of

drugs and estrogen rings A and B, 1-naphthyl-4(5)- imidazole, also inhibited microsomal estrogen-16- hydroxylation. The results of this study indicate that insecticide synergists may affect the breakdown of estrogenic hormones in the organism.

76-2339. Benedek, P. (P. O. Box 127, H-1502 Budapest, Hungary). **Effect of night temperature on the toxicity of field-weathered mevinphos residues to honeybees.** *Z. Angew. Entomol.* 79(3): 328-331; 1975. (4 references)

Studies were conducted to determine the influence of various night temperatures on the toxicity of field-weathered residues of mevinphos to honeybees. Phosdrin was sprayed on experimental plots at the dosage of 1.5 kg/ha. Results of the studies showed that the toxicity of mevinphos residues to honeybees was adversely affected by air temperature, as rises in temperature caused sharp declines in bee mortality. At low temperature mevinphos residues caused almost total bee mortality in both 8 and 12 hr samples. However, their toxicity became moderate at 12-13°C. These findings suggest that temperature is the key factor in relation to bee mortality from mevinphos plant residues. Accordingly, the chemical should not be applied anytime in blooming winter rape fields as the changeable weather and low average temperatures on spring evenings would be hazardous to the bees.

76-2340. Sazonov, P. V. (Author address not given). **Osnovnye napravleniya v formirovanii assortimenta khimicheskikh sredstv. [Basic directions in the developmental patterns of an assortment of organic pesticides.]** *Zashch. Rast. (Moscow)* 9: 20-21; 1974. (Russian)

The basic trends in the development of the assortment of pesticides in the USSR are outlined. The development of new preparations with reduced toxicity to warm-blooded animals has been the most characteristic trend during the last decade. Most modern pesticides have LD50 values ranging from 200 to 1,000 mg/kg, while the LD50 of tedion (tetradifon) and neoron (bromopropylate) is over 5,000 mg/kg. Along with the development of low-toxicity preparations, highly persistent pesticides, such as DDT, polychloropinene (strobane), and polychlorocamphene (toxaphene), will be phased out gradually. The combination of chemical and biological means of pest control is made possible by the development of preparations with very low toxicity in entomophagous species as compared with their toxicity for the pests to be controlled, e.g., rogor (dimethoate), phosalone, menazon, and phthalophos (phosmet). Pest control with preparations pertaining to different classes is expected to prevent the manifestation or reduce the resistance of pests to pesticides.

76-2341. Andruszewska, A. (Inst. Biol. Roslin AR, Szczecin, Poland). **Badania nad wplywem herbicydow stosowanych w lesnictwi na grzyby glebowe ze szczegolnym uwzglednieniem grzybow mikoryzowych. [Studies on the effect of herbicides used in forestry on soil fungi especially mycorrhiza.]** *Zesz. Nauk. Akad. Roln. Szczecinie* 10(39): 3-21; 1973. (22 references) (Polish)

Alipur (cycloron plus chlorbufam), Chwastox Liquid 30 (MCPA), gramoxone (paraquat), and simazine were applied under experimental field conditions and effect on pine and spruce seedlings and mycorrhiza in roots was determined. In addition, the effect of different concentrations of these herbicides on the mycelia of mycorrhiza and spore formation of several soil fungi was examined in vitro. Alipur (6 l/ha in 400 l water) and gramoxone (4 l/ha in 400 l water) had a deleterious effect on the seedlings and on mycorrhiza and in the 0.5-2.0% concentration were quite toxic in vitro. Chwastox (5 l/ha in 600 l water) had no significant effect on mycorrhiza but in 0.5-1.17% concentration killed all fungi in vitro; in 0.17% concentration it stimulated the growth of *Armillaria mellea*. Simazine (4 kg/ha in 400 l water) had no effect on mycorrhiza and seedlings and was the least toxic in vitro (in 0.5-2.0% concentration) and stimulated the growth of *Fusarium oxysporum* and *Agaricus campestris*.

76-2342. Jakubowska, B.; Nowak, A. (Inst. Soil Sci., Fertil., Szczecin, Poland). **Wplyw niektorych insektycydow fosfoorganicznych i karbaminianow na wzrost grzybow glebowych. [Effect of some organophosphorus and carbamate insecticides on growth of soil fungi.]** *Zesz. Nauk. Akad. Roln. Szczecinie* 10(39): 141-150; 1973. (7 references) (Polish)

Acetone solutions of propoxur, carbaryl, gardona (tetrachlorvinphos), and DDVP (dichlorvos) were added to Sabouraud's medium in 10, 30, 60, and 120 ppm amounts and the effects on size and morphology of colonies of a number of soil fungi were determined over a 21 day period. In most cases a dose related inhibitory effect was noted whereby the organophosphorus compounds caused a more pronounced inhibition than the carbamate insecticides. The strongest inhibition was noted with DDVP followed by gardona, carbaryl, and propoxur (in that order) except for *Sepedonium chrysospermum* which was strongly inhibited by all 4 compounds and *Geotrichum candidum* which was more sensitive to gardona than to DDVP. The insecticides had a growth stimulating effect on *Fusarium javanicum*, *Mucor fragilis*, *Mucor hiemalis* (after carbaryl) and *Penicillium aurantio-candidum* (after gardona) or showed no effect (*Geotrichum candidum* after DDVP, carbaryl, and propoxur). Some species (*Mycelia sterilia*, *Geotrichum candidum*, *Sepedonium chrysospermum*, *Fusarium javanicum*, *Fusarium sambucinum*) reacted with morphologic changes in the appearance of their colonies (white, more fluffy surface or low, less dense growth).

76-2343. Gevorkyan, S. G.; Movsesyan, N. A.; Topchyan, A. A. (Armenian Branch, All-Union Scientific Res. Inst. Hyg. Toxicol., USSR). **K toksikologicheskoy otsenke**

gerbitsida mezoranila. [On the toxicological evaluation of the herbicide Mezoranil.] *Zh. Eksp. Klin. Med. Akad. Nauk Armyanskoy SSR* 15(5): 32-36; 1975. **(Russian)**

The chronic and acute toxicity of a new herbicide, mezoranil (aziprotryn) was studied in rats, mice, and rabbits. According to the manufacturer (Ciba-Geigy, Switzerland), the oral LD50 in rats is 5,833 mg/kg, and over 3,000 mg/kg on skin application. The acute oral LD50 was found to be 3,696 mg/kg in rats and 635 mg/kg in mice. Venous plethora, focal hemorrhages into the cerebral membranes and parenchymatous membranes, and dystrophic and necrotic changes were observed in animals poisoned with high doses. The threshold dose, determined on the basis of the ability of the central nervous system to summate subthreshold impulses, is 300 mg/kg. A weak cumulative property was established in the chronic experiment (4 months).

see also **76-2134 76-2168 76-2185 76-2357**

76-2344. Wilson, A. J. (Environ. Protection Agency, Gulf Breeze Environ. Res. Lab., Sabine Island, Gulf Breeze, FL). **Effects of suspended material on measurement of DDT in estuarine water.** *Bull. Environ. Contam. Toxicol.* 15(5): 515-521; 1976. (30 references)

Samples of estuarine and distilled water were fortified with 3.0 ppm *p,p'*-DDT in acetone, extracted with petroleum ether, ethyl ether in hexane, or methylene chloride, and analyzed using electron capture gas chromatography. Immediately after addition of DDT to the estuarine water, all solvent systems removed 93% of the pesticide. Recovery from the distilled water was similar after 14 days incubation, but the recoveries from estuarine water at that time were reduced 49% and 28% with petroleum ether and methylene chloride, respectively. Failure of the solvents to remove DDT sorbed onto particulate matter in the estuarine water may have accounted for the reduced recovery after incubation. In a second study, fortified estuarine water and fortified artificial estuarine water containing an algal culture were extracted with methylene chloride. In some cases, the suspended material in the water was filtered out prior to extraction and analyzed separately from the filtrate. In all tests, there was a significant increase in the recovery of DDT when the suspended material was analyzed separately. The greatest increase was in the fortified artificial estuarine water containing the algal culture. The results indicate that data obtained by analysis of water using methods which do not efficiently extract sorbed pollutants from suspended material may be misleading.

76-2345. Yamashita, Y.; Suzuki, Y.; Tomaru, K.; Kaburaki, Y. (Author address not given). **[Studies of pesticide residues on tobacco (1). Analytical method for organochlorine pesticides.]** *Nippon Sembai Kosha Chuo Kenkyusho Kenkyu Hohoku (Sci. Pap. Central Res. Inst. Jpn. Monopoly Corp.)* 115: 15-22; 1973. (17 references) (Japanese)

Analytical methods were studied for detecting residues of organochlorine pesticides in cured tobacco leaves. The results of the study indicated that there was no significant difference in efficiency for such determinations between petroleum ether and *n*-hexane, which were used for liquid partition and the subsequent chromatographic cleanup of the leaf extracts. Therefore *n*-hexane can be used instead of petroleum ether for cleanup procedures of the leaf extracts. The pesticides were chromatographically separated into 2 groups by Florisil column using 4 and 15% ethyl ether in *n*-hexane as the eluting solvent. The groups were: A (α-BHC, β-BHC, γBHC, δ-BHC, aldrin, heptachlor, heptachlor epoxide, *p,p'*-DDE, *o,p'*-TDE, *o,p'*-DDT, *p,p'*-TDE, and *p,p'*-DDT) and B (dieldrin and endrin). Several kinds of GC-column liquid phase on Anakrom ABS were examined for the separation of the pesticides. QF-1(7.5%) DC-200(5%) (7:5) were suitable as the liquid phase. Under these conditions, 12 pesticides and their related compounds of A group and 2 compounds of B group were separated in single injection performance, respectively. Recoveries of the pesticides and variations of values obtained by this procedure were sufficient for the analysis of organochlorine pesticide residues in cured tobacco leaves.

76-2346. Mesaki, T.; Tsuge, S.; Suzuki, K.; Kashiwa, T. (Gumma Prefect. Inst. Environ. Pollut., Maebashi, Gumma, Japan). **[A gas-liquid chromatographic determination of CVMP dust formulation (2-chloro-1(2',4',5'-trichlorophenyl) vinyl dimethyl phosphate.]** *Noyaku Kensasho Hokoku (Bull. Agric. Chem. Inspect. Stn.)* 15: 19-21; 1975. (Japanese)

A simple and rapid method of determining 2-chloro- 1(2',4',5'-trichlorophenyl) vinyl phosphate in a powder formulation was presented. The powder formulation was extracted with acetone and the extract was concentrated under reduced pressure. The concentration was subjected to gas-liquid chromatography on a column packed with SP 2401 GasChrom Q using diacetoxymethylnaphthalene as internal standard. The recovery from freshly formulated pesticide was 99.9%, and the accuracy of determination of the commercial formulation was shown by the S.D. of 0.013.

76-2347. Sakamoto, T.; Kashiwa, T. (Agric. Chem. Inspect. Stn., Minist. Agric. For., Kodaira, Tokyo, Japan). **[Determination of ethylene thiourea in ethylenebisdithiocarbamate funcidal formulations.]** *Noyaku Kenshasho Hokoku (Bull. Agric. Chem. Inspect. Stn.)* 15: 31-35; 1975. (8 references) (Japanese)

Ethylenethiourea which may be contained as a degradation product in ethylenebisdithiocarbamate fungicide formulation, was studied by TLC and GC. The methanol extract of a formulation sample was placed on a silica gel TLC plate, and was developed with a 5:1 mixture of ethyl acetate and benzene. To prevent decomposition, in the case of amobam the extract was diluted with a 2:8 mixture of diethylamine and methanol before placement on the TLC plate and then developed with a 10:1:1 mixture of ethyl acetate, acetone, and diethylamine. The detection limit for ethylenethiourea was 0.3 μg/spot in case of coloring with potassium ferricyanate and ferric chloride, and 1 μg/spot for UV detection. Formulations of amobam, zineb, and maneb, and technical grades of methylam and polycarbamate were found to contain more than 0.2% ethylenethiourea while mancozeb contained less than 0.2 but more than 0.07% of ethylenethiourea. Gas chromatography was applied to the methanol extract using poymeric beads of 2,6-diphenyl-*p*-phenylene oxide as column packing and a hydrogen flame in the detector.

76-2348. Lakshminarayana, V.; Rengarajan, T. (Cent. Plant Protection Training Inst., Hyderabad 30, India). **Thin-layer clean-up and subsequent determination of ethyl parathion and methyl parathion in their formulations.** *Res. Ind.* 20(3): 153-155; 1975. (2 references)

A convenient and rapid method is presented based on thin layer clean-up of parathion or methyl parathion formulations and their subsequent determination via yellow sodium nitrophenate. The results obtained with this technique are in close agreement with those obtained by conventional methods. The TLC clean-up technique has the advantage of eliminating the washing of the sample with dilute, chilled sodium carbonate, thus avoiding the chance of a part of the active ingredient being washed out. The TLC clean-up method is useful in determining the shelf-life of parathion and methyl parathion formulations. It is very easy to separate by TLC *p*-nitrophenol, which is the decomposition product of the insecticide. Therefore, the extent of degradation of the formulation can be rapidly determined. The TLC clean-up method is also cheaper and very rapid compared to conventional methods. One estimation can be completed in about 1.5 hr, compared to the conventional 3 hr.

76-2349. Nakamura, T. (Res. Lab., Sankyo Co. Ltd., Tokyo, Japan). **Hymexazol, a new plant protecting agent. IV. Metabolism and residues. 4. Residue in plants and soils.** *Sankyo Kenkyusho Nenpo (Ann. Rep. Sankyo Res. Lab.)* 25: 45-47; 1973. (4 references)

A specific gas chromatographic method was developed for determining residues of hymexazol in plants and soils. The method is based on derivatization to thiophosphate after removing interfering substances. The residue levels were determined in rice grains and grapes and in soils under both laboratory and field conditions at different intervals after hymexazol applications. The diethylthiophosphoryl derivative formed was detected specifically at the nanogram level with phosphorus sensitive detectors using 3% OV-17 on chromosorb W or SE-42 on chromosorb G as the stationary phase. Minimum detectable amounts were about 1 ng with a flame photometric detector and 0.2 ng with a flame thermionic detector. Hymexazol was extracted with acetone. The minimum detectable level was about 0.02 ppm in a flame photometric detector and 0.005 ppm in a flame thermionic detector on the basis of a sample size of 100 g. In actual analyses, hymexazol (600 ppm) was applied 1 or 2 times at a rate of 3 l/m^2 to soils for the control of damping-off in rice seedlings. No hymexazol was detected in the harvested grains. When hymexazol was sprayed twice at a rate of 150 l/10A to rice plants about 2 months prior to harvest, the residues were negligible. However, when an aqueous solution of hymexazol (600 ppm) was applied several times in midsummer at a rate of 250 l/10A to grapes, slight residues were found in fruits sampled at different intervals after the final application. The glucosides into which hymexazol is metabolized in plants were hardly detected by this method of analysis. In laboratory testing, hymexazol was incorporated into wet soils and submerged soils at a rate of 120 ppm based on dry weight of soil. In field tests, with hymexazol applied twice at a total dose of 90 ppm, the persistence was fairly long in Shiga clay loam, but short in other soils. The approximate half lives of hymexazol under both lab and field conditions were 20 and 25 days in Shiga clay loam and 10 and 2 days in other soils. In addition to microbial degradation, irreversible adsorption to soil components such as organic matter and clay minerals must be assumed to be one of the main factors concerned with the persistence of hymexazol in soils.

76-2350. Gorbacheva, N. A.; Smirnov, V. M. (Sci. Res. Inst. Forensic Med., USSR Ministry of Health, USSR). **Ob izbiratel'nosti obnaruzheniya fosfamida (rogora) pri sudebno-khimicheskom issledovanii trupnogo materiala. [Selectivity of phosphamide (rogor) detection in forensic chemical analyses of cadaver materials.]** *Sud.-Med. Ekspert.* 18(4): 29-32; 1975. (Russian)

The selectivity and specificity was studied of a thin-layer chromatographic method for the determination of dimethoate (rogor, phosphamide). Following extraction with acidulated water and subsequent extraction with chloroform, the sample was applied on silicic acid layer in benzene, and development was done with a hexane-dioxane mixture at a ratio of 1:1. In the case of cadaver materials, it was possible to detect dimethoate in the presence of DEF, tetrachlorvinphos, diazinon, chlorpyrifos, coumaphos, methyl parathion, fenitrothion, menazon, ronnel, phosmet, phosalone, and crotoxyphos. Additional reactions made it possible to detect dimethoate in the presence of trichlorfon and technical grade methylmercaptophos and naled. Quantitative isolation from technical preparations of formothion and malathion was not possible.

76-2351. Pomerantsev, V. I.; Bruk, A. A.; Gosteva, N. A. (Emergency Hospital No. 10 of the Leningrad Municipal Health Department, Leningrad, USSR). **Kolichestvennoe opredelenie dikhloretana v biologicheskikh sredakh metodom gazo-zhidkostnoy khromatografii. [Quantitative determination of dichloroethane in biological materials by gas-liquid chromatography.]** *Sud.-Med. Ekspert.* 18(4): 32-34; 1975. (Russian)

A gas-liquid chromatographic method is described for the quantitative determination of dichloroethane in biological materials such as blood and urine. Gas chromatograph with flame-ionization detector, 80-100 mesh chromosorb P as column filling with 10% 1,2,3-*tris*-2-cyanoethoxypropane, a column temperature of 140°C, an evaporator temperature of 160°C, and a nitrogen flow rate of 35 ml/min are used for the determination. Quantitative determination is done by means of a calibration curve as a function of peak height or peak area. The error of the determination is 5% (relative). The limit of detection is 0.1 mg%. Different linear relationships were found for the concentration and the peak area or peak height for blood and urine.

76-2352. Krylova, A. N. (Sci. Res. Inst. Forensic Med., USSR Ministry of Health, Moscow, USSR).

Ekstraktsionno-fotometricheskoe opredelenie olova v morinom v biologicheskom materiale. [Extraction photometric determination of tin with morin in biological material.] *Sud.-Med. Ekspert.* 18(4): 48-51; 1975. (Russian)

A method is described for the colorimetric determination of tin in biological material through complex formation with morin. Human liver and kidney with artificially increased tin content were used for the experiments. After mineralization of the sample with sulfuric or nitric acid over 3-4 hours, avoiding the precipitation of stannic oxide, tin complex of morin is formed in a pH range of 0.8-1.2. The optimal morin concentration is 0.1%. The complex is extracted with a mixture of ethylacetate and carbon tetrachloride. The method has sufficient accuracy for the determination of tin concentrations as occur usually in poisoning cases.

76-2353. Gazaryan, V. S. (Erevan Institute of Animal Husbandry and Veterinary Science, Erevan, USSR). **Ekspress-metod opredeleniya medi v patologicheskom materiale. [Express method for determining copper in pathological material.]** *Veterinariya (Moscow)* 1: 95-96; 1976. (Russian)

An express method is described for the determination of copper in pathological tissues and organs. The sample, e.g., the liver of sheep, is dried and pulverized for incineration at 700-750°C, after which the ash is dissolved in distilled water. After filtration and neutralization with 1% hydrochloric acid, the filtrate is combined with 20% freshly prepared potassium ferrocyanide. The presence of copper is indicated by light pink to dark brick red coloration. Copper contents of 70-140 mg% were determined in liver samples from sheep chronically poisoned with contaminated hay from vineyards that had been treated with blue vitriol. No copper was determined in the liver of normal sheep.

76-2354. Yaroshenko, V. I. (Ukr. Sci. Res. Inst. Exp. Vet. Med., Karkov, USSR). **Viznachennya khlorofosu v medu enzimatichnim agar-difuznim metodom. [Determination of chlorophos in honey by an enzymic agar-diffusion method.]** *Visn. Sil'skogospod. Nauki* 1: 108-111; 1974. (9 references) (Ukrainian)

When incorrectly used, many pesticides will have toxic effects on beneficial insects, warm-blooded animals, and human beings. The greatest danger to bees is from improper use of chemicals in agriculture. The influence of pesticides may reach man through the consumption of honey. There are several methods for determination of residues in honey. Experiments were conducted to determine residues of organophosphorus compounds by the Sandi agar-diffusion method as modified by A. A. Nepoklonova and V. K. Metelytsia. In this method chlorophos (trichlorfon) diffuses into agar containing cholinesterase and bromthymol blue as indicator, and inhibits the enzyme action. In preparations of agar with an aqueous solution of acetylcholine, degradation of the latter occurs, yielding choline and acetic acid. The acid changes the pH of the medium, and thus the indicator agar changes to a greenish or yellow color. The presence of chlorophos is characterized by the appearance of blue circles around an indentation in the agar. The experiments conducted suggest high sensitivity of the agar-diffusion method, which may be used for determining chlorophos content in honey.

76-2355. Sackmauerova, M.; Madaric, A.; Palusova, O. (Nat. Inst. Hyg., Bratislava, Czechoslovakia). **Stanovenie chlorovanych insekticidov v rybach. [Determination of chlorinated insecticides in fish.]** *Vod. Hospod.* 24(5): 135-137; 1974. (9 references) (Slovak)

Numerous incidents of fish kills due to pesticide pollution of rivers and lakes have given rise to a need for methods of determining pesticides in fish tissues. The tissues are extracted with warm petroleum ether and cleaned up by petroleum ether-acetonitrile partitioning. The extract is further cleaned up on a sulfuric acid-treated Celite column. The eluate is spotted onto Silufol plates for thin-layer chromatography. It is possible to separate *p,p'*-DDD (TDE), *p,p'*-DDE, α-BHC, and heptachlor by gas chromatography on a column containing 3-5% SE-30 on chromosorb W, and to separate the four BHC isomers, *o,p'*-DDT, *p,p'*-DDT, *p,p'*-DDE, and *p,p'*-DDD on a column containing 1.5% OV-17 plus 2% QF-1 on Chromosorb W. Additional reliability is attained by comparing the results of gas chromatographic separation on the two columns.

76-2356. Gillespie, D. M.; Eldredge, J. D.; Thompson, C. K. (Dep. Biol., Univ. Utah, Salt Lake City, UT 84112). **A kinetic model for static bioassay of insecticides.** *Water Res.* 9(9): 817-819; 1975. (9 references)

Static bioassays, although widely used in evaluating the effects of toxins in aquatic environments, provide rather imprecise data, especially when used with relatively insoluble toxins. A mathematical model of such a system was developed, assuming only first order chemical kinetics. The model was tested using DDT as toxin and stoneflies (*Pteronareys californica* Newport) as assay organisms. The data suggested that DDT was lost from the system at a rapid rate. Fifty-six percent of the DDT was not recovered immediately after its addition, and this was attributed to the hydrophobic nature of DDT. Monitoring the DDT concentrations in test and control systems and fitting the data to the proposed model permitted calculation of the mean toxin dosage per insect, thereby yielding relatively precise and useful toxicological information. When the constants for such a system are determined, they can be applied to future bioassays in similar systems.

76-2357. Wendel, K. G. (Dep. Bot. Mycol., Univ. Essen, Essen, Germany). **Rhizosphaerenorganismen unter dem Einfluss pestizider Umwelt-Belastungen. I. Ein Beitrag zur Frage des Fungizidnachweises (TMTD = Tetramethylthiuramdisulfid) in Boeden nach Modell-Versuchen.**

[Rhizosphere organisms as affected by environmental contamination with insecticides. I. Detection of a fungicide (TMTD = tetramethylthiuram disulfide) in soils in model experiments.] *Zentralbl. Bakteriol. Parasitenkd. Infektionskr. Hyg. Abt.* Orig. Reihe A 228(1-2): 141-153; 1974. (42 references) (German)

Methodological problems and results of biological tests are described for the detection of thiram in soil in model experiments. Thiram was detected by a microbiological inhibition test using *Aspergillus niger*. The growth inhibition was different in different soils as a function of the humus content, i.e., of the intensity of the degradation of the fungicide. The fungicidal effect was lowest after 3-4 months, after which a rebound was observed due to the fungicidal activity of the metabolites of thiram.

see also 76-2188 76-2278

PESTICIDES ABSTRACTS

QUARTERLY INDEX

JULY–SEPTEMBER 1976 Vol. 9, Nos. 7–9

PREFACE

The basic design for this index was supplied by the Technical Services Division, Office of Pesticide Programs, Environmental Protection Agency. It consists of two subject indexes (concept and compound) and two author indexes (personal and corporate). Primary and secondary headings in the Concept Index appear as subordinate headings in the Compound Index, both indexes having three levels of specificity.

In the Concept Index, only general aspects are indexed under the broader terms shown below. For specific terms, the reader should consult the appropriate heading in the Concept Index, selecting the narrower term that best fits his need.

Compounds are indexed by their common names; where other compound designations are used by authors, these synonyms appear with the notation to *see* the common name. Articles referring, in general, to structural or functional chemical classes are indexed under the broader headings (e.g., organochlorines, herbicides, etc.).

Alternative controls
Analysis
Beneficial effects
Biochemical effects (*see also* Enzyme activity)
Blood/body fluids
Carcinogenesis
Cardiovascular system
Cytological effects
Digestive system
Economics
Endocrine system (*see also* Reproduction/growth)
Environmental pollution
Enzyme activity (*see also* Biochemical effects)
Excretory system
Experimental design
Factors influencing metabolism/toxicity
Immunology
Integumentary system
Laws and regulations
Metabolism (*see also* Residue degradation)
Morbidity and mortality statistics
Musculoskeletal system
Mutagenesis/teratogenesis (*see also* Cytological effects)
Nervous system
Nomenclature
Photodecomposition (*see also* Residue degradation)
Poison control centers
Prevention
Reproduction/growth (*see also* Endocrine system)
Residue degradation (*see also* Photodecomposition *and* Metabolism)
Residue dynamics
Residue removal
Residues/air
Residues/non-target organisms
Residues/food and feed
Residues/human
Residues/plants
Residues/soil
Residues/water
Respiratory system
Reticuloendothelial system
Reviews
Safety standards
Sensory system
Therapeutic use
Toxicity/non-target organisms
Toxicity/experimental animals
Toxicity/humans
Treatment of poisoning

Subject Index: Concepts

Absorption
see also Metabolism
Chicken
Carbaryl, 76-2320
Cow
Ronnel, 76-2319
Fish
Toxaphene, 76-2234
In vitro
Aldrin, 76-1962
Chlordimeform, 76-1962
DDT, 76-1962
Dieldrin, 76-1962
Organochlorines, 76-1981
Parathion, 76-1962
Pig
Carbaryl, 76-1812
Phosmet, 76-1812
Trichlorfon, 76-1812
Plankton/algae
2,4-D, 76-1748
Plants
Fenitrothion, 76-1970
Rat
Paraquat, 76-1754

Adrenal
see also Endocrine system
Animals/experimental
Carbaryl, 76-1790
Coumaphos, 76-1790
Dichlorvos, 76-1968
Heptachlor, 76-1951
Lindane, 76-1968
Ronnel, 76-1951
Trichlorfon, 76-1792
Human
TDE, 76-1765

Alternative controls, 76-1594, 76-1855 76-2103, 76-2104

Amino acids/peptides/proteins
see also Biochemical effects
Animals/experimental
Altosid, 76-1736
Altozar, 76-1736
BHC, 76-1819
Carbaryl, 76-1763
Chlordane, 76-2280
DDT isomers, 76-1996
Ethylene dibromide, 76-2047
PMA, 76-2301
Thiram, 76-2006
Tri-*o*-cresyl phosphate, 76-2052
Trichlorfon, 76-1789, 76-1794
Human
Organophosphates, 76-1924
Pyrazon, 76-1923
Thiram, 76-2006
In vitro
Chlorfenvinphos derived compound, 76-1727
DDT derived compounds 76-2285

Amino acids/peptides/proteins (cont'd)
Dichlorvos, 76-2058
Microorganisms
Captan, 76-2291
Dieldrin, 76-2291

Analysis
see also Bioassay; Chromatography; Electrometry; Enzyme assay; Spectrometry
General, 76-2095
Fungicides, 76-2068
Herbicides, 76-2069
Juvenile hormones and analogs 76-2066
Organophosphates, 76-2067
Pyrethrins, 76-2067
Sample preparation
Ancymidol, 76-2070, 76-2070
Atrazine, 76-2094
Benomyl, 76-1845
Carbamates, 76-1846
Daminozide, 76-2070
DDE, 76-2094
Diazinon, 76-2062
Dichlorvos, 76-2063
Dieldrin, 76-2094
Dimethoate, 76-2101
Fenitrothion, 76-2062
Herbicides, 76-2070
Hexaflurate, 76-1851
Methyl parathion, 76-2348
Organochlorines, 76-1846 76-2345
Organophosphates, 76-1846
Parathion, 76-2084, 76-2348
Phorate, 76-2062
Protect, 76-2070
Thiabendazole, 76-1845
Thiophanate, 76-1845

Behavior
see also Nervous system
Animals/experimental
Benomyl, 76-2266
Carbendazim, 76-2266
Dieldrin, 76-2050
Herbicides, 76-2232
Trichlorfon, 76-1793
Trichlorophos, 76-1804

Beneficial effects
Herbicides, 76-2113

Bibliographies, 76-2112

Bioassay
see also Analysis
DDT, 76-2356
Ezomycin A, 76-1837
Ezomycin B, 76-1837
Simazine, 76-1850
Thiram, 76-2357

Biochemical effects
see also Amino acids/peptides/proteins; Carbohydrates; Catecholamines; Electrolytes; Enzyme activity; Lipids/steroids/sterols; Nitrogen cycle; Nucleic acids; Porphyrins/pigments; Respiration, cellular; Vitamins/coenzymes
General
Chlorfenvinphos, 76-2248
DDT, 76-1760, 76-2048
Dieldrin, 76-2048
Dioxins, 76-1784
Famphur, 76-1771
Nabam, 76-1947
Thiram, 76-1947
Trichlorfon, 76-2323
Zineb, 76-1947
Microorganisms
Herbicides, 76-2326

Biotransformation
see also Metabolism 76-1782
General
DDT, 76-1760
Annelids
Aldrin, 76-2287
Chicken
DDT, 76-1782
Methoprene, 76-2025
Cow
BHC, 76-1782
DDT, 76-1782
Dog
Tebuthiuron, 76-2051
Duck
Tebuthiuron, 76-2051
Fish
Aldrin, 76-1985
Tebuthiuron, 76-2051
Fungi
Carboxin, 76-1820
Fungicides, 76-1977
In vitro
Carbaryl, 76-2283, 76-2284
Chlorfenvinphos derived compound, 76-1727
Chlortoluron, 76-2283, 76-2284
HEOM, 76-2282
IDIMP, 76-1972
Methabenzthiazuron, 76-1745
Methyl parathion, 76-2281
Mirex, 76-1898
Parathion, 76-1728
Phosphamidon, 76-2283, 76-2284
Insects
Dicofol, 76-1720
HEOM, 76-2282
Leptophos, 76-2278, 76-2278
Monocrotophos, 76-2278, 76-2278
Parathion, 76-2278, 76-2278
Phospholan, 76-2278
Triphenyl phosphate, 76-1955

Biotransformation (cont'd)
Microorganisms
BAY NTN 9306, 76-2022
Carbamates, 76-2036
Dichloran, 76-2019
Dieldrin, 76-2055
Endosulfan, 76-2055
Fungicides, 76-2036
Glyphosate, 76-1816
Herbicides, 76-2064
Isopropalin, 76-2333
Lindane, 76-2055
Linuron, 76-1976
Methylmercury dicyandiamide 76-1743
Organochlorines, 76-2230
Organophosphates, 76-2030
Phorate, 76-1984
Piperonylate, 76-2033
Polychlorinated biphenyls 76-1762
Monkey
Mirex, 76-2243
Pig
Carbaryl, 76-1812
Chloronaphthalenes, 76-2018
Phosmet, 76-1812
Trichlorfon, 76-1812
Plankton/algae
Chlorpyrifos, 76-2237
Plants
Amitrole, 76-2004
Azinphosmethyl, 76-2015
BAY NTN 9306, 76-2022
Bentazon, 76-1817, 76-2334 76-2335
Carbamates, 76-2241
Carbofuran, 76-2038
Chlorpropham, 76-2020
Cyanazine, 76-2336
2,4-D, 76-1943
Dicamba, 76-2014
Endosulfan, 76-2023
Ethephon, 76-1717
Ethion, 76-2023
Ethylene thiourea, 76-2021
Fenitrothion, 76-1970
Herbicides, 76-1715
Isopropalin, 76-1815, 76-2333
Lindane, 76-1978, 76-2035
Malathion, 76-2032
Maneb, 76-2021
Methabenzthiazuron, 76-1814
Metribuzin, 76-1818, 76-1980
Nabam, 76-2021
Organochlorines, 76-2241
Organophosphates, 76-2241
Zineb, 76-2021
Rabbit
Tebuthiuron, 76-2051
Rat
DDT isomers, 76-2000
Dinoseb, 76-1946
Lindane, 76-1729, 76-2247
Malathion, 76-1710
Phosmet, 76-2329
Squid

Biotransformation (cont'd)
DFP, 76-2043

Blood/body fluids
see also Blood cells; Lymph; Plasma/serum
Animals/experimental
Phosmet, 76-2327
Thiram, 76-2005

Blood cells
see also Blood/body fluids
Animals/experimental, 76-2290
Famphur, 76-1771
Lindane, 76-2293
Parathion, 76-2302
Trichlorfon, 76-1789, 76-1803
Trichlorophos, 76-1803, 76-1803
Human
Chlordane, 76-1935
In vitro
DDT, 76-2264

Brain
see also Nervous system
Animals/experimental
DDT isomers, 76-1996
Dichlorvos, 76-2269, 76-2271
Trichlorfon, 76-2269, 76-2270 76-2271

Carbohydrates
see also Biochemical effects
Animals/experimental, 76-2268
BHC, 76-1819
Chlordane, 76-2235, 76-2280
Malathion, 76-1710, 76-2331
Trichlorfon, 76-1795, 76-2270
Human
DDT, 76-1753
In vitro
Fenaminosulf, 76-1938

Carcinogenesis
General
Atrazine, 76-1725
Carbamates, 76-2049
DDT, 76-1675
Fungicides, 76-2049
Animals/experimental
Carbaryl, 76-2239, 76-2240
Dieldrin, 76-2303
Organochlorines, 76-1761
Human
Nitro compounds, 76-2219
Organochlorines, 76-1761 76-1868
Microorganisms
Chloroxuron, 76-2256
Dimefox, 76-2256
Thiram, 76-2256

Cardiovascular system
see also Heart
Human
Thallium, 76-2218

Catecholamines
see also Biochemical effects
Animals/non-target
Heptachlor, 76-1951, 76-1951

Catecholamines (cont'd)
Ronnel, 76-1951

Cell membranes
see also Cytological effects
Animals/experimental
Malathion, 76-1710
Microorganisms
2,4-D, 76-1748

Chromatography
see also Analysis
General
2,4,5 T derived compound 76-1690
Column, 76-2060
Dimethoate, 76-2101
Organochlorines, 76-1839 76-2073
Polychlorinated biphenyls 76-1839, 76-2073
Gas-liquid, 76-1834, 76-1835 76-2096
Allethrin, 76-2081
Ancymidol, 76-2070
Atrazine, 76-2094
BHC isomers, 76-2071
Carbamates, 76-1826, 76-2078 76-2087
Cyperquat, 76-2093
2,4-D, 76-2088, 76-2091
Daminozide, 76-2070
DDE, 76-2094
DDT, 76-2071, 76-2100, 76-2344
Diazinon, 76-2062
Dieldrin, 76-2071, 76-2094
Ethylene dichloride, 76-2351
Ethylene thiourea, 76-2347
Fenitrothion, 76-2062
Fungicides, 76-2087
Glyphosate, 76-2080
Heptachlor, 76-2355
Herbicides, 76-1826, 76-2070 76-2078, 76-2085
Hexachlorobenzene, 76-2071
Hymexazol, 76-2349
Malathion, 76-2090
Malathion derived compounds 76-2090
Methamidophos, 76-2097
Methoprene, 76-2092
Nitro compounds, 76-2085
Organochlorines, 76-1844 76-2345
Organophosphates, 76-1844 76-2079, 76-2085
Oryzalin, 76-2089
Parathion, 76-2084, 76-2097
PCNB, 76-1838, 76-2071
Phorate, 76-2062
Polychlorinated biphenyls 76-2100
Propanil, 76-1848
Protect, 76-2070
Pyrethrin I, 76-2081
Tetrachlorvinphos, 76-2346
Gel permeation
Malathion, 76-1830

Chromatography (cont'd)
Parathion, 76-1830
Paper
Organophosphates, 76-1827
Thin-layer
BHC isomers, 76-2355
Carbamates, 76-2065, 76-2082
Carbaryl, 76-1828
DDT, 76-2355
Dimethoate, 76-2101, 76-2350
Ethylene thiourea, 76-2347
Famphur, 76-2099
Fungicides, 76-2065
Methyl parathion, 76-2074 76-2348
Nitro compounds, 76-1842
Organochlorines, 76-1833 76-2098
Organophosphates, 76-1842 76-1847, 76-2082
Paraquat, 76-2083
Parathion, 76-1836, 76-2061 76-2348
Parathion derived compounds 76-2075

Chromosomes/genes
see also Cytological effects
Animals/experimental
DDE, 76-2263
DDT, 76-2263
TDE, 76-2263
Tepa, 76-1770
Human
Chemosterilants, 76-1974
Fungicides, 76-1974
Herbicides, 76-1974
In vitro
2,4-D, 76-1786
DDT, 76-2264
Plants
Ethyl mercuric chloride, 76-1787
Maleic hydrazide, 76-1735

Cytological effects
see also Cell membranes; Chromosomes/genes; Fibroblasts; Mitochondria; Mitosis/meiosis

Animals/experimental
Carbaryl, 76-2251
Soman, 76-1952
Plants
Carbaryl, 76-2251

Demyelination
see also Nervous system
Human
Trichlorfon, 76-1686

Digestive glands
see also Digestive system
Animals/experimental
Chlorfenvinphos, 76-2248

Digestive glands (cont'd)
Ronnel, 76-1805
Trichlorfon, 76-2315
Trichloro-metaphos-3, 76-1805

Digestive system
see Digestive glands; Liver; Pancreas (exocrine)

Distribution/storage
see also Metabolism
General
DDT, 76-1760
Annelids
Aldrin, 76-2287
Birds
Oxadiazon, 76-1714
Chicken
Carbaryl, 76-2320
Granosan, 76-2328
Methoprene, 76-2024, 76-2025
Cow
Oxadiazon, 76-1714
Ronnel, 76-2319
Trichlorfon, 76-1795
Fish, 76-2330
DDT, 76-2330
Human
DDE, 76-1967
DDT isomers, 76-1967
Monkey
Fenitrothion, 76-1650
Mirex, 76-2242, 76-2243
Mouse
Hexachlorobenzene, 76-1776
PCNB, 76-1776
Pheasant
Lindane, 76-2037
Mercurials, 76-1723
Pig
Carbaryl, 76-1812
Granosan, 76-1806
Phosmet, 76-1812
Trichlorfon, 76-1812
Plankton/algae
Naled, 76-2236
Plants
Bentazon, 76-1817
Methabenzthiazuron, 76-1814
Pronamide, 76-2332
Rabbit
Trichlorfon, 76-2323
Rat
Hexachlorobenzene, 76-1776 76-1999
PCNB, 76-1776
PCP, 76-1966
Phosmet, 76-2329
PMA, 76-2301
Sheep
Lindane, 76-1796
Turkey
Ethion, 76-2031

Economics
Carbamates, 76-1598
Organochlorines, 76-1598

Economics (cont'd)
Organophosphates, 76-1598

EEG
see also Nervous system
Animals/experimental
Dieldrin, 76-1779
Lindane, 76-2244
Sarin, 76-1779
Human, 76-2207
Carbamates, 76-1687
Organophosphates, 76-1687 76-2225, 76-2227

Eggshell effects
see also Reproduction/growth
Animals/experimental
DDT isomers, 76-1721

Electrolytes
see also Biochemical effects
Animals/experimental
Parathion, 76-1711
Human
Organophosphates, 76-1682

Electrometry
see also Analysis
Polarography
Benefin, 76-2064
Dinitramine, 76-2064
Isopropalin, 76-2064
Nitralin, 76-2064
Oryzalin, 76-2064
PCNB, 76-1838
Trifluralin, 76-2064

Embryo/fetus
see also Reproduction/growth
Animals/experimental, 76-1758
DDT, 76-1969
DDT isomers, 76-1969
Lindane, 76-1949, 76-2037
Human, 76-1758
Plants
Fenitrothion, 76-1970

Endocrine system
see also Adrenal; Gonads; Pancreas (endocrine)
Animals/experimental
DDT, 76-2013
Lindane, 76-2013

Environmental pollution, 76-1600 76-1604, 76-1858, 76-2114
Carbamates, 76-2108
DDT, 76-1601, 76-1606
Fungicides, 76-1854, 76-2108
Herbicides, 76-2113
Mercurials, 76-1605, 76-1606 76-2105, 76-2106
Organochlorines, 76-1602, 76-2102 76-2108, 76-2109
Organophosphates, 76-1602, 76-2102 76-2108
Polychlorinated biphenyls, 76-1601 76-2102
Pyrethrins, 76-1602
Synergists, 76-1602

Enzyme activity
see also Biochemical effects
76-1782, 76-1782
General
BHC isomers, 76-1733
Captan, 76-1732
Carbaryl, 76-1773
DDT, 76-1718, 76-1733
Fumigants, 76-1808, 76-1808
Granosan, 76-1806
Herbicides, 76-1808
Organochlorines, 76-1808
76-1808, 76-1979, 76-1981
Organophosphates, 76-1752
76-1808
Paraquat, 76-1712
Polychlorinated biphenyls
76-1733
Trichlorfon, 76-1773, 76-1795
Acid phosphatase
Chlordane, 76-2280
Dichlorvos, 76-2271
Ovex, 76-1995
Petkolin, 76-1995
Phosalone, 76-1995
Trichlorfon, 76-2271, 76-2315
Aliesterase
Dichlorvos, 76-1971
Alkaline phosphatase
Chlordane, 76-2280
Dichlorvos, 76-2271
Malathion, 76-1994
ATPase, 76-2268
Trichlorfon, 76-2270
Cholinesterase, 76-1789, 76-2196
Antidotes, 76-1988
Armine, 76-2002
Azinphosmethyl, 76-1950
BPMC derived compounds
76-1730
Carbaryl, 76-1772
Chlorfenvinphos, 76-2248
Chlorothion, 76-1931
CKB 1028, 76-1940
Demephion, 76-1940
DFP, 76-1942
Dichlorvos, 76-1971, 76-2271
Dimethoate, 76-1940
Famphur, 76-1771
Fenitrothion derived compound
76-2288
Heptachlor, 76-1951
IDIMP, 76-1972
Leptophos, 76-1777
Malathion, 76-1931
Methyl demeton, 76-2208
Methyl parathion, 76-1940
Monocrotophos, 76-1719
Organophosphates, 76-1764
76-1936, 76-1941, 76-2206
Paraoxon, 76-1942
Parathion, 76-1929, 76-1931
76-2302
Phosmet, 76-2327
Propham, 76-1772
Ronnel, 76-1951
Sarin, 76-1942

Enzyme activity (cont'd)
Soman, 76-1952
Temophos, 76-1998
Trichlorfon, 76-1731, 76-1793
76-1803, 76-1940, 76-2271
Trichlorophos, 76-1803
Cytochrome oxidase
Dichlorvos, 76-2269, 76-2272
Trichlorfon, 76-2269, 76-2272
DNase
Rotenone, 76-2010
Esterases
Carbaryl, 76-2283, 76-2284
Chlortoluron, 76-2283, 76-2284
CKB 1028, 76-1940
Demephion, 76-1940
Dimethoate, 76-1940
Hexachlorobenzene, 76-1781
Malathion, 76-1775, 76-1781
76-1955
Methyl parathion, 76-1940
Phosphamidon, 76-2283, 76-2284
Trichlorfon, 76-1940
Triphenyl phosphate, 76-1955
β-Glucuronidase
Trichlorfon, 76-2315
GOT
Carbaryl, 76-1772
Propham, 76-1772
6-GPD
Malathion, 76-2331
GPT
Carbaryl, 76-1772
Propham, 76-1772
Pyrazon, 76-1923
Invertase
Atrazine, 76-2029
Lactic dehydrogenase
Malathion, 76-2331
Pyrazon, 76-1923
Mixed function oxidases
Aldrin, 76-1724, 76-1985
DDT, 76-1753, 76-1760, 76-2317
Dieldrin, 76-1724
Lindane, 76-1729
Mirex, 76-2286
Organochlorines, 76-2059
Organophosphates, 76-2059
76-2298
Parathion, 76-1728, 76-1945
PMA, 76-2301
Synergists, 76-2338
Triarimol, 76-1780
NADH dehydrogenase
Fenaminosulf, 76-1938
Oximes
DFP, 76-1971
Polygalacuronase
Atrazine, 76-2029
RNase
Rotenone, 76-2010
Succinic dehydrogenase
Dichlorvos, 76-2269, 76-2272
Malathion, 76-1994
Trichlorfon, 76-2269, 76-2272
UDP-glucuronyl transferase
Organophosphates, 76-1961

Enzyme assay
see also Analysis
Cholinesterase, 76-2060, 76-2077
Trichlorfon, 76-2354

Estrogens
see also Reproduction/growth
In vitro
Synergists, 76-2338

Excretion
see also Metabolism
Chicken
Methoprene, 76-2024
Cow
Organochlorines, 76-1766
Dog
Tebuthiuron, 76-2051
Duck
Tebuthiuron, 76-2051
Fish
Tebuthiuron, 76-2051
Insects
Phospholan, 76-2278
Monkey
Mirex, 76-2242
Rabbit
Tebuthiuron, 76-2051
Rat
DDT isomers, 76-2000
PCP, 76-1966
Tebuthiuron, 76-2051
Sheep
Carbaryl, 76-1811
Trichlorfon, 76-1811

Excretory system
see Kidney

Experimental design
Epidemiology, prevention, and treatment, 76-2216
Toxicology and pharmacology
76-1758, 76-1800
Chloroform, 76-2313
Organochlorines, 76-1757
76-1798
Organophosphates, 76-1798

Factors influencing metabolism/toxicity
General
Paraquat, 76-1783
Adaptation
Chemosterilants, 76-1821
2,4-D, 76-1990
Juvenile hormones and analogs
76-1821
MCPA, 76-1990
Methylmercury dicyandiamide
76-1743
Molinate, 76-1990
Ovex, 76-1995
Petkolin, 76-1995
Phosalone, 76-1995
Warfarin, 76-2011
Age, 76-2330
Bentazon, 76-2335
DDT, 76-2330
Dieldrin, 76-2303
Biological magnification

Factors influencing metabolism/toxicity (cont'd)
DDT, 76-1809
Electroshock
Chlordane, 76-1774
Formulation
DDT, 76-2304
Fenitrothion, 76-2304
Thiram, 76-2005
Geographic location
Warfarin, 76-2011
Humidity
Malathion, 76-2032
Monocrotophos, 76-1719
Picloram, 76-1907
Interactions
BHC, 76-2316
BHC isomers, 76-1733
Chlordane, 76-1774
DDT, 76-1718, 76-1733, 76-1903 76-2304
Dieldrin, 76-2053
Fenitrothion, 76-2304, 76-2316
Hexachlorobenzene, 76-1781
Lindane, 76-1729
Malathion, 76-1781
Organochlorines, 76-1766 76-1975
Paraquat, 76-1712, 76-1767
Parathion, 76-2053
Polychlorinated biphenyls 76-1733, 76-2316
Potassium-2-formyl- 1,4-chlorophenoxyacetate, 76-2316
Tepa, 76-1770
Trichlorfon, 76-1731
Light/radiation
Malathion, 76-1994
Methbenzthiazuron, 76-1745
Nutritional state
Chloroform, 76-2310
DDT, 76-2048, 76-2317
Dieldrin, 76-2048
Paraquat, 76-1767
pH
Chloroform, 76-2310
Route
Cystaphos, 76-1769
Schedule of dosage
Atrazine, 76-2040
Carbaryl, 76-2320
Cyanazine, 76-2040
2,4-D, 76-2040
Ethylene thiourea, 76-2306
Ronnel, 76-2319
Simazine, 76-2040
Season
Lindane, 76-2035
Sex
Azinphosmethyl, 76-1950
Mevinphos, 76-2318
Mirex, 76-2286
Structure/function
DDE, 76-1948
DDT isomers, 76-1948
Herbicides, 76-2321
Mercurials, 76-2295

Factors influencing metabolism/toxicity (cont'd)
Organochlorines, 76-2321
Organophosphates, 76-1941
Taxon
Bentazon, 76-2335
BHC, 76-2316
2,4-D, 76-1749, 76-1990
DDT, 76-1718, 76-1969
DDT isomers, 76-1969
DFP, 76-1971
Dichlorvos, 76-1971, 76-1971
Fenitrothion, 76-2316
Herbicides, 76-1715, 76-2326
MCPA, 76-1990
Molinate, 76-1990
Phorate, 76-2289
Polychlorinated biphenyls 76-2316
Potassium-2-formyl- 1,4-chlorophenoxyacetate, 76-2316
Temperature
Lindane, 76-1734
Mevinphos, 76-2339
Picloram, 76-1907

Fertility/sterility
see also Reproduction/growth
Animals/experimental
Dichlorvos, 76-1964
Lindane, 76-1949
Malathion, 76-1964

Fibroblasts
see also Cytological effects
Animals/experimental
BHC, 76-2314
Carbaryl, 76-2314
DDT, 76-2314
Thiram, 76-2314
Zineb, 76-2314
Human
BHC, 76-2314
Carbaryl, 76-2314
DDT, 76-2314
Thiram, 76-2314
Zineb, 76-2314
In vitro
Dichlorvos, 76-2058
Trichlorfon, 76-2057

Gametogenesis
see also Reproduction/growth
Animals/experimental
Ethylene dibromide, 76-2047
Microorganisms
Captan, 76-2291

Gonads
see also Endocrine system
Animals/experimental
DDT, 76-1969
DDT isomers, 76-1969

Growth
see also Reproduction/growth
Animals/experimental, 76-1758 76-2290
Azinphosmethyl, 76-1950
Heptachlor, 76-1951

Growth (cont'd)
Lindane, 76-1949
Ronnel, 76-1951
Rotenone, 76-2322
Trichlorfon, 76-1793
Human, 76-1758
Microorganisms
Atrazine, 76-2029
BHC, 76-2299
Carbaryl, 76-1944, 76-1993 76-2342
Chloroform, 76-2311, 76-2312
Chlorpyrifos, 76-2237
Cyanazine, 76-2041
2,4-D, 76-2041
DDE, 76-2299
DDT, 76-1944, 76-1993, 76-2299
Dichlorvos, 76-2342
Dimethoate, 76-1944
Diquat, 76-2265
Heavy metals, 76-2299
Heptachlor, 76-2299
Herbicides, 76-2341
Hexachlorobenzene, 76-2299
Malathion, 76-1944
Menazon, 76-1944
Organochlorines, 76-1808
Organophosphates, 76-1808
PMA, 76-2238
Polychlorinated biphenyls 76-2299
Propoxur, 76-2342
Rotenone, 76-2322
Simazine, 76-2041
Temophos, 76-1993
Tetrachlorvinphos, 76-2342
Plants, 76-2341

Heart
see also Cardiovascular system
Human
Organophosphates, 76-1682
Parathion, 76-2222, 76-2223

Hormones
see also Lipids/steroids/sterols; Reproduction/growth
Animals/experimental
Dieldrin, 76-2053
Parathion, 76-2053

Immunology
Animals/experimental
Tri-*o*-cresyl phosphate, 76-2052
In vitro
DDT, 76-2001
Fungicides, 76-2001
Maleic anhydride, 76-2001

Integument
see Skin

Kidney
see also Excretory system
Animals/experimental
DDT, 76-1939
DDT isomers, 76-1996
Hexachlorobenzene, 76-1939
Methylacetono-chlorophos-3 76-1807
Organophosphates, 76-1939

Kidney (cont'd)
Schradan, 76-1939
Soman, 76-1952
2,4,5-T, 76-1778
Thallium, 76-1939
Thiram, 76-2005
Trichlorfon, 76-1792
Warfarin, 76-1939
Animals/non-target
Granosan, 76-1806

Laws and regulations
FAO, 76-1920
Germany (BRD), 76-2215
Nitro compounds, 76-2219
Japan
Organochlorines, 76-1694
Organophosphates, 76-1694
Mexico, 76-1692
Spain
DDT, 76-1918
Organophosphates, 76-1918
United Kingdom
Nitro compounds, 76-2219
USA, 76-1919
DDT, 76-1675
Nitro compounds, 76-2219
World Health Organization, 76-1920

Lipids/steroids/sterols
see also Biochemical effects; Hormones
Animals/experimental
BHC, 76-1819
Carbaryl, 76-1790, 76-1791
Coumaphos, 76-1790, 76-1791
Organochlorines, 76-1987
Paraoxon, 76-2046
Paraquat, 76-1767
Parathion, 76-2046
Human
Chlordecone, 76-2045
DDE, 76-1967
DDT isomers, 76-1967
Parathion, 76-1929

Liver
see also Digestive system
Animals/experimental
BHC, 76-1819
BHC isomers, 76-1823
Carbaryl, 76-1763, 76-1791
Copper sulfate, 76-2008
Coumaphos, 76-1791
DDT, 76-1760, 76-1782, 76-1939 76-1983
Dieldrin, 76-1782, 76-2053, 76-2303
Dinoseb, 76-1946
Dioxins, 76-1784
Heptachlor, 76-1939, 76-1951
Hexachlorobenzene, 76-1939 76-1999
Methylacetono-chlorophos-3 76-1807
Mixed function oxidases, 76-1782
Nabam, 76-1947
Organophosphates, 76-1939
Parathion, 76-2053
Parathion derived compound

Liver (cont'd)
Parathion derived compound 76-1957
PMA, 76-2301
Ronnel, 76-1951
Schradan, 76-1939
Thallium, 76-1939
Thiram, 76-1947, 76-2005
Triarimol, 76-1780
Trichlorfon, 76-1792
Warfarin, 76-1939
Animals/non-target
Granosan, 76-1806
Human
DDT, 76-1760
Pyrazon, 76-1923
In vitro
Synergists, 76-2338

Lung
see also Respiratory system
Animals/experimental
Paraquat, 76-1713, 76-1744 76-1747, 76-1754, 76-1783
Human
Paraquat, 76-1927, 76-1932 76-2197

Lymph
see also Blood/body fluids
Animals/experimental, 76-2290

Metabolism
see also Absorption; Biotransformation; Distribution/storage; Excretion
Amphibians
DDT, 76-1960
Cow
Arsenicals, 76-2054
Dichlorvos, 76-1797
Difluron, 76-2028
Trichlorfon, 76-1797
Hamster
DDT, 76-2337
Helminths
Phorate, 76-2289
Human
Arsenicals, 76-2054
In vitro
Fenitrothion derived compound 76-2288
Microorganisms
Chloroxuron, 76-2256
Cycluron, 76-2256
Dimefox, 76-2256
Organochlorines, 76-1986
Thiram, 76-2256
Mouse
DDT, 76-1741, 76-2337
Validamycin, 76-2276
Rat
Carbamates, 76-2049
Carbaryl, 76-1722
Chlordane, 76-1722
Fungicides, 76-2049
Leptophos, 76-1722
Parathion, 76-1722
2,4,5-T, 76-1778

Metabolism (cont'd)
Sheep
Chlorpyrifos-methyl, 76-2034
Ferbam, 76-2026

Microsomes
In vitro
Aldrin, 76-1724
Dieldrin, 76-1724

Mitochondria
Plants
Captan, 76-1716
2,4-DB, 76-1716

Mitosis/meiosis
see also Cytological effects
In vitro
Benomyl, 76-2042
Carbendazim, 76-2042
Lindane, 76-2042
Rotenone, 76-2042
Thiram, 76-2042
Microorganisms
Dichlorvos, 76-2258
Fungicides, 76-2255
Herbicides, 76-2257

Morbidity and mortality statistics
Argentina, 76-1673, 76-1674
Hungary
Arsenicals, 76-1678
Organophosphates, 76-2205
Japan, 76-1701
Mercurials, 76-1680
Mexico
Organochlorines, 76-1693
Organophosphates, 76-1693
Netherlands, 76-2202
Sweden
Mercurials, 76-1680
USSR, 76-2217

Muscle, striated
see also Musculoskeletal system
Animals/experimental
2,4-D, 76-1739
DDT, 76-2296
In vitro
Armine, 76-2002

Musculoskeletal system
see also Muscle, striated
Animals/experimental
Trichlorfon, 76-1792

Mutagenesis/teratogenesis
General
Carbamates, 76-2049
Fungicides, 76-2049
Animals/experimental, 76-2228 76-2229
Amitrole, 76-2252
Benomyl, 76-2245
Carbamates, 76-2305
DDE, 76-2263
DDT, 76-2263
Dichlorvos, 76-2261
Ethylene thiourea, 76-2250 76-2253, 76-2306, 76-2307
Ethylmercury *p*-toluene sulfona-

Mutagenesis/teratogenesis (cont'd)
mide, 76-2260
Fenitrothion derived compound 76-2288
Fungicides, 76-2305
Hexachlorobenzene, 76-1776
Hexachlorophene, 76-1751
PCNB, 76-1776
TDE, 76-2263
Tepa, 76-1770
Animals/non-target
2,4-D, 76-1953
2,4,5-T, 76-1953
Human
Chemosterilants, 76-1974
Fungicides, 76-1974
Herbicides, 76-1974
In vitro
Benomyl, 76-2245
DDT, 76-2264
Microorganisms, 76-2259
Amitrole, 76-2252
Captan, 76-2262
Carbaryl, 76-2267
Carbaryl derived compound 76-2016
Diazinon, 76-2016
Dichlorvos, 76-2249
Dodine, 76-2267
Ethylene thiourea, 76-2250
Fumigants, 76-1808
Fungicides, 76-1808, 76-2254 76-2257
Herbicides, 76-1808, 76-2254 76-2257
Linuron, 76-2016
Organochlorines, 76-1808 76-2254
Organophosphates, 76-1808
Prometryne, 76-2267
Plants
Maleic hydrazide, 76-1735

Nervous system
see also Behavior; Brain; EEG; Peripheral nerves; Spinal cord; Sympathetic nerves; Thermoregulation
Animals/experimental, 76-2306
Leptophos, 76-1777
Methylacetono-chlorophos-3 76-1807
Human
Arsenicals, 76-2213
Chlordecone, 76-1684, 76-1926 76-2045
Organophosphates, 76-1924

Nitrogen cycle
see also Biochemical effects
Microorganisms
Atrazine, 76-2294
Chloroform, 76-2309, 76-2310 76-2311, 76-2312
2,4-D, 76-1956
Diquat, 76-2265

Nitrogen fixation (cont'd)
Disulfoton, 76-2292
Fenitrothion, 76-2308
Malathion, 76-2308
Phorate, 76-2308
Simazine, 76-2294

Nucleic acids
see also Biochemical effects
Animals/experimental
Altosid, 76-1736
Altozar, 76-1736
BHC isomers, 76-1823
Butonate, 76-2279
Carbaryl, 76-1763
Trichlorfon, 76-2279
Microorganisms
Captan, 76-2291
Dieldrin, 76-2291

Pancreas (endocrine)
see also Endocrine system
Animals/experimental
Chlordane, 76-2235

Pancreas (exocrine)
see also Digestive system
Animals/experimental
Trichlorfon, 76-2315

Peripheral nerves
see also Nervous system
Animals/experimental
DFP, 76-2043
PMA, 76-2273
Human
Aldicarb, 76-2203
Benomyl, 76-2203
DDT, 76-2203
Dichlorvos, 76-2203

Photodecomposition, 76-2143
Atrazine, 76-1725
Carbaryl, 76-1891
2,4-D, 76-1902
DDT, 76-1884
Herbicides, 76-1866
Inezin, 76-2126
Menazon, 76-1893
Organophosphates, 76-1612
Phoxim, 76-1859
Thiabendazole, 76-1875
Triforine, 76-1639

Placental transfer
see also Reproduction/growth
Human
BHC, 76-1649
DDT derived compounds 76-1649

Plasma/serum
see also Blood/body fluids
Animals/experimental
Trichlorfon, 76-1794, 76-1803
Human
DDE, 76-1967
DDT isomers, 76-1967
Organophosphates, 76-1924
Pralidoxime, 76-1958
In vitro

Plasma/serum (cont'd)
DDT derived compounds 76-2285

Poison control centers
Netherlands, 76-2202

Polyneuritis
Human
Trichlorfon, 76-1686

Porphyrins/pigments
see also Biochemical effects
Animals/experimental
Dinoseb, 76-1946
Hexachlorobenzene, 76-1999
Human
Thallium, 76-2210
In vitro
Parathion, 76-1728
Plants
Captan, 76-1716
2,4-DB, 76-1716

Prevention
Decontamination
Lindane, 76-1937
Methyl mercaptophos, 76-1937
Disposal, 76-2214, 76-2215
Organochlorines, 76-1925
Organophosphates, 76-1925
Education/training, 76-2198
Protective equipment, 76-1934
Storage and transportation, 76-1917 76-2216

Reproduction/growth
see also Eggshell effects; Embryo/fetus; Estrogens; Fertility/sterility; Gametogenesis; Growth; Hormones; Placental transfer; Reproductive organs, male
Animals/experimental
Dieldrin, 76-1802, 76-1997

Reproductive organs, male
see also Reproduction/growth
Animals/experimental
Benomyl, 76-2246

Residue degradation
General
Mercurials, 76-2127
Naphthaleneacetic acid, 76-2187
Food and feed
Aldicarb, 76-1882
Thiram, 76-1911
Plants, 76-1613
Atrazine, 76-1668
Dicamba, 76-1880
Dimethoate, 76-1888
Malathion, 76-1888
PCNB, 76-2185
Picloram, 76-1880
Soil, 76-1671

Residue degradation (cont'd)
Aldrin, 76-1617
Atrazine, 76-1668, 76-2164
Benefin, 76-2167
BHC, 76-2178
BPMC, 76-2188
Carbamates, 76-2189
Carbaryl, 76-1671
Carbofuran, 76-2129
Clonitralide, 76-1613
CNP, 76-2183
2,4-D, 76-1672
DDT, 76-1617, 76-2129, 76-2178
Dicamba, 76-1880
Dieldrin, 76-1617
Disulfoton, 76-1908
Diuron, 76-2164
Endosulfan, 76-1617
Fenitrothion, 76-2129
Fensulfothion, 76-2129
Fungicides, 76-1644
Glyphosate, 76-2168
Herbicides, 76-1644, 76-2132
Linuron, 76-2164
Maleic hydrazide, 76-2163
MSMA, 76-2170
Nitralin, 76-2167
Organochlorines, 76-1644
PCNB, 76-2184
Perfluidone, 76-2171
Picloram, 76-1880
Propyzamide, 76-1665, 76-2133
Simazine, 76-1652, 76-2164
Tebufos, 76-1883
Thiram, 76-1878
Trifluralin, 76-2167
Water
Atrazine, 76-2149
Carbaryl, 76-2149
2,4-D, 76-2149
DDT, 76-2159
Desmetryne, 76-2149
Lindane, 76-2124
Malathion, 76-1909, 76-2149
MCPA, 76-2149
Organochlorines, 76-2130
Parathion, 76-1909, 76-2124 76-2149
Polychlorinated biphenyls 76-2159
Ro 10-3108, 76-1900
2,4,5-T, 76-2149

Residue dynamics
Fenitrothion, 76-2123
Herbicides, 76-1622
Organophosphates, 76-1612
Parathion, 76-2121
2,4,5-T, 76-1645

Residue removal
Food and feed
DDT, 76-1901, 76-2161
Granosan, 76-2161
Lindane, 76-2161
Plants
Parathion, 76-1871
Water

Residue removal (cont'd)
DDT, 76-2122
Dieldrin, 76-2157
Endrin, 76-2122
Heptachlor, 76-2122
Heptachlor epoxide, 76-2157
Lindane, 76-2157
Malathion, 76-2122

Residues/air
Industrial
Fungicides, 76-2173
Mercurials, 76-2173
Organophosphates, 76-2115
PCP, 76-1633

Residues/food and feed
General
DDT, 76-1885
Diazinon, 76-2131
Hexachlorobenzene, 76-2151
Mercurials, 76-2181
Organochlorines, 76-1625 76-1670, 76-1916, 76-2180 76-2181, 76-2194, 76-2195
Organophosphates, 76-1670 76-2181
Polychlorinated biphenyls 76-1625
Pyrethrins, 76-1661
Total diet, 76-1635
BHC, 76-1614
BHC isomers, 76-1874
DDT, 76-1614
DDT isomers, 76-1874
Dieldrin, 76-1614
Lead, 76-1614
Mercurials, 76-1614
Organochlorines, 76-2158
Polychlorinated biphenyls 76-1614
Animal feed
BHC isomers, 76-2154
DDE, 76-1865, 76-1876, 76-2154
DDT, 76-1865, 76-1903, 76-2154
DDT isomers, 76-1876
Dieldrin, 76-1865, 76-2154
Endrin, 76-2154
Hexachlorobenzene, 76-1876 76-1894
Lindane, 76-1865
Organochlorines, 76-1904
Polychlorinated biphenyls 76-1876
TDE, 76-2154
Baby formula
DDT, 76-2120
Cereals
Herbicides, 76-1658
TCA, 76-1618
Dairy products
Arsenicals, 76-2054
BHC, 76-1610, 76-2135
BHC isomers, 76-1653, 76-1915 76-2142
Carbaryl, 76-1663
DDE, 76-1653, 76-1876, 76-2142
DDT, 76-1653, 76-1663, 76-1915

Residues/food and feed (cont'd)
DDT, 76-2135, 76-2142
DDT isomers, 76-1876
Dieldrin, 76-1610
Hexachlorobenzene, 76-1876
Lindane, 76-1663
Methyl parathion, 76-1663
Organochlorines, 76-1646 76-1664, 76-1669, 76-1869 76-1886, 76-1904
Polychlorinated biphenyls 76-1876
TDE, 76-1653
Fish
BHC, 76-2160
DDT, 76-2160
Mercurials, 76-2119
Fruits
Arsenicals, 76-1862
BHC, 76-1656
Carbaryl, 76-1609, 76-1914
Chlorthalonil, 76-2192
DDE, 76-1656
DDT, 76-1656, 76-2175
Diazinon, 76-1627
Fenitrothion, 76-2123, 76-2182
Fungicides, 76-1662
Lead, 76-2191
Monocrotophos, 76-1892
Organochlorines, 76-1864
Oxythioquinox, 76-2193
Parathion, 76-2118
Silvex, 76-1873
Meat
Arsenicals, 76-2054
BHC, 76-1860
DDT, 76-1860
Lindane, 76-1860
Organophosphates, 76-1860
Poultry
DDE, 76-1876
DDT isomers, 76-1876
Hexachlorobenzene, 76-1876
Polychlorinated biphenyls 76-1876
Vegetables
Aldicarb, 76-1879, 76-1882
Arsenicals, 76-2054
Captan, 76-2186
Carbofuran, 76-1896
Chlordane, 76-1881
Cyanophos, 76-2179
DDT, 76-2175
Diazinon, 76-1627
Dimethoate, 76-1888
Disulfoton, 76-1879
Ethion, 76-1896
Fensulfothion, 76-1896
Herbicides, 76-1658
Lead, 76-2191
Malathion, 76-1888
Maneb, 76-2190
Organophosphates, 76-1864
PCNB, 76-2184
Phorate, 76-1896
Terbufos, 76-1620

Residues/humans
General
BHC, 76-1648
DDT, 76-1648
Dieldrin, 76-1648
Organochlorines, 76-1916
Adipose
BHC, 76-1651
DDE, 76-1651
DDT, 76-1651
DDT isomers, 76-1870
Hexachlorobenzene, 76-2151
Organochlorines, 76-1611 76-1868
Polychlorinated biphenyls 76-1870
Blood
BHC, 76-1641, 76-1642, 76-1643 76-1649
DDT, 76-1641, 76-1642, 76-1643
DDT derived compounds 76-1649
Organochlorines, 76-1868
Milk, 76-1651
BHC, 76-1647
BHC isomers, 76-1874, 76-1913
DDE, 76-1647, 76-1651
DDT, 76-1647, 76-1651, 76-1861 76-1913
DDT isomers, 76-1874
Organochlorines, 76-1615 76-1886, 76-1904, 76-2180 76-2194, 76-2195
Polychlorinated biphenyls 76-1640, 76-1913
Urine
Arsenicals, 76-2054
BHC isomers, 76-2174
Fungicides, 76-2173
Mercurials, 76-2173

Residues/non-target organisms
General
DDT, 76-1809
Mercurials, 76-2127
Mirex, 76-1630, 76-1632
Birds
Mercurials, 76-1626, 76-2117
Organochlorines, 76-2117
Crustacea
DDT, 76-2145
Dieldrin, 76-1638
Hexachlorobenzene, 76-2116
Hexachlorobutadiene, 76-2116
Organochlorines, 76-1631
Parathion, 76-2121
Duck
Cadmium, 76-1634
DDE, 76-1637
DDT, 76-1634, 76-1637
Dieldrin, 76-1634, 76-1637
Hexachlorobenzene, 76-1634
Lead, 76-1634
Mercurials, 76-1634
Polychlorinated biphenyls 76-1634, 76-1637
TDE, 76-1637
Fish

Residues/non-target organisms (cont'd)
BHC, 76-1657
BHC isomers, 76-1912
DDT, 76-1636, 76-1657, 76-1912 76-2139
Dieldrin, 76-1636, 76-1638
Hexachlorobenzene, 76-2116
Hexachlorobutadiene, 76-2116
Mercurials, 76-1636, 76-2139
Organochlorines, 76-1631 76-1872
Organophosphates, 76-1657
Polychlorinated biphenyls 76-1636, 76-1912, 76-2139
Insects
Dieldrin, 76-1638
Microorganisms
DDT, 76-2137
Molluscs
Organochlorines, 76-1631 76-1872
Monkey
Fenitrothion, 76-1650
Plankton/algae
DDT, 76-2145
Plants
Fenitrothion, 76-2123
Seal
DDT, 76-2150
Dieldrin, 76-2150
Mercurials, 76-2150
Polychlorinated biphenyls 76-2150

Residues/plants
Cotton
Arsenicals, 76-2054
Forest
Dicamba, 76-2141
Phosphamidon, 76-1616
Picloram, 76-2141
2,4,5-T, 76-1645
Medicinals and condiments, 76-1867
Dimethoate, 76-1888
Endosulfan, 76-1867
Malathion, 76-1888
Organochlorines, 76-1863
Organophosphates, 76-1863
Phoxim, 76-1859
Orchard
Benomyl, 76-2134
Carbendazim, 76-2134
Thiophanate-methyl, 76-2134
Ornamentals
Organochlorines, 76-1863
Organophosphates, 76-1863
Paper
BHC, 76-2152

Residues/soil
General
Atrazine, 76-1668
Fungicides, 76-1644
Herbicides, 76-1644, 76-1659 76-1866
Mercurials, 76-2181
Methyl bromide, 76-1623
Mirex, 76-1632

Residues/soil (cont'd)
Organochlorines, 76-1644 76-2181
Organophosphates, 76-2181
Adsorption
Arsenicals, 76-1660
Atrazine, 76-2172
Carbaryl, 76-2155
DDT, 76-2137, 76-2138
Dieldrin, 76-2138
Glyphosate, 76-1816, 76-2168
Heptachlor, 76-2138
Herbicides, 76-2153
Maleic hydrazide, 76-2163
Metribuzin, 76-1667
Picloram, 76-2156
Pronamide, 76-2332
Pyrazon, 76-2162
Secbumeton, 76-2172
Simazine, 76-1652
Telone, 76-1654
Terbutryne, 76-2172
Movement, 76-1671, 76-2148
Arsenicals, 76-1660
BHC, 76-2177, 76-2178
Carbaryl, 76-1671, 76-2155
Carbofuran, 76-2129
DDT, 76-2129, 76-2138, 76-2177 76-2178
1,2-dibromoethane, 76-1887
Dicamba, 76-2141
1,3-dichloropropene, 76-1887
Dieldrin, 76-2138
Fenitrothion, 76-2129
Fensulfothion, 76-2129
Glyphosate, 76-1816, 76-2168
Heptachlor, 76-2138
Perfluidone, 76-2171
Picloram, 76-1624, 76-2141
Prometryne, 76-1666, 76-2169
Simazine, 76-1652
Volatilization
2,4-D, 76-2165
DDT, 76-1619
Dieldrin, 76-1899
Heptachlor, 76-1899
Lindane, 76-2124
Parathion, 76-2118, 76-2124

Residues/water
General
Atrazine, 76-1629
DDE, 76-1629
Dieldrin, 76-1629
Hexachlorobenzene, 76-2151
Organochlorines, 76-2130
Groundwater/rain, 76-2147
Aldrin, 76-2146
BHC, 76-2146
2,4-D, 76-1906
DDE, 76-2146
DDT, 76-2146
Dieldrin, 76-2146
Herbicides, 76-2153
Lindane, 76-2146
Irrigation
2,4-D, 76-1655
TCA, 76-2166

Residues/water (cont'd)
Lakes/ponds
BHC, 76-1657
DDT, 76-1657
Organochlorines, 76-2176
Organophosphates, 76-1657
Oceans/seas
DDE, 76-1877
DDT, 76-1877, 76-2139
Mercurials, 76-2139
Polychlorinated biphenyls 76-1877, 76-2139
TDE, 76-1877
Rivers/streams, 76-2144
Arsenicals, 76-2136
Chlordane, 76-2140
2,4-D, 76-2125
DDT, 76-2139, 76-2140
Dichlorvos, 76-1628
Dieldrin, 76-1638
Hexachlorobenzene, 76-2116
Hexachlorobutadiene, 76-2116
Lead, 76-2136
Lindane, 76-2140
Mercurials, 76-2139
Polychlorinated biphenyls 76-2139, 76-2140

Respiration, cellular
see also Biochemical effects
Animals/experimental
DDT, 76-1983, 76-2056
Endosulfan, 76-2056
Trichlorfon, 76-2270
In vitro, 76-1726
Carbaryl, 76-2283, 76-2284
Chlortoluron, 76-2283, 76-2284
Organochlorines, 76-1726
Organophosphates, 76-1726
Parathion, 76-1945
PCP, 76-1954
Phosphamidon, 76-2284
Microorganisms
2-Chlorobenzamide, 76-1742
Chloroform, 76-2309, 76-2312
Dazomet, 76-1742
Fumigants, 76-1808
Fungicides, 76-1808
Herbicides, 76-1808
Nitrapyrin, 76-1742
Organochlorines, 76-1808
Organophosphates, 76-1808
PMA, 76-2238
Tributyl-3-chlorobenzylammonium bromide, 76-1742
Plants
Captan, 76-1716
2,4-DB, 76-1716

Respiratory system
see also Lung; Upper respiratory tract
Human
Mercurials, 76-1699
Organochlorines, 76-1699
Organophosphates, 76-1699
Parathion, 76-2222

Respiratory system (cont'd)
Tin compounds, 76-1699

Reviews
Epidemiology, prevention, and treatment, 76-1921
Monitoring and residues, 76-2128
BHC, 76-1857
DDT, 76-1857
Fungicides, 76-2107
Herbicides, 76-2107
Mercurials, 76-2110
Organochlorines, 76-2107
Polychlorinated biphenyls 76-1857
Simazine, 76-1910
Toxicology and pharmacology 76-2300
Endrin, 76-2231
Mercurials, 76-2110
Organochlorines, 76-1987

Safety standards
Acceptable daily intake
Organochlorines, 76-1694
Organophosphates, 76-1694
OSHA, 76-2204
TLV/MAC, 76-2204
Tolerances, 76-1917, 76-1920
Toxicity rating class, 76-2204

Sensory system
see Vision

Skin
see also Integument
Animals/experimental
Carbamates, 76-2275
Dichlorvos, 76-1959, 76-2233
Fungicides, 76-2275
Trichlorophos, 76-1804
Human
Arsenicals, 76-1678
Pyrazon, 76-1923
2,4,5 T derived compound 76-1690
Thiophanate-methyl, 76-1707
Zineb, 76-1930

Spectrometry
see also Analysis
Atomic absorption/emission
Arsenicals, 76-1824
Naled, 76-1840
Colorimetry
Carbaryl, 76-1831, 76-1832
Copper, 76-2353
DDT derived compounds 76-1825
Dichlorvos, 76-1852
Fentin acetate, 76-1843
Organophosphates, 76-1849
Paraquat, 76-1841
Parathion, 76-1829
Polychlorinated biphenyls 76-1825
Tin compounds, 76-2352
Trichlorfon, 76-2354
Infrared
Atrazine, 76-1725

Spectrometry (cont'd)
PCNB, 76-1838
Mass spectrometry
Atrazine, 76-1725
Glyphosate, 76-2080
NMR
Atrazine, 76-1725
Radiometry, 76-2086
UV
Atrazine, 76-1725
Benomyl, 76-1845
DDT derived compounds 76-1825
Dicumarol, 76-2076
Polychlorinated biphenyls 76-1825
Thiabendazole, 76-1845, 76-2072
Thiophanate, 76-1845
Warfarin, 76-2076

Spinal cord
see also Nervous system
Animals/experimental
Leptophos, 76-2044
In vitro, 76-1726
Organochlorines, 76-1726
Organophosphates, 76-1726

Structure/function
Factors influencing metabolism/toxicity
Mercurials, 76-2295

Sympathetic nerves
see also Nervous system
Animals/experimental
Phosmet, 76-2327
In vitro
Armine, 76-2002

Therapeutic use
Cancer
Maleic hydrazide, 76-2027
Piperonyl butoxide, 76-2253
Rotenone, 76-2010
Cushing's syndrome
TDE, 76-1759, 76-1765
Demodicidosis
Dichlorvos, 76-1959
Muscular dystrophy, 76-2003

Thermoregulation
see also Nervous system
Animals/experimental
Carbaryl, 76-1785, 76-1785
DDT, 76-1785, 76-1785
Parathion, 76-1785, 76-1785

Thymus
Animals/experimental
Dioxins, 76-1784

Toxicity/experimental animals
General, 76-1740, 76-2340
Mercurials, 76-2295
Naled, 76-1738
Bee
Dimethoate, 76-1755
Fenitrothion, 76-2297
Fenthion, 76-2297
Herbicides, 76-1756

Toxicity/experimental animals (cont'd)
Birds
Carbamates, 76-1801
Fungicides, 76-1801
Herbicides, 76-1801
Naled, 76-1738
Organochlorines, 76-1801
Organophosphates, 76-1801
Trichlorfon, 76-1813
Chicken
Atrazine, 76-2017
Phosmet, 76-2327
Trichlorfon, 76-1803
Trichlorophos, 76-1803
Crustacea
Dieldrin, 76-2039
Endothall, 76-2039
Malathion, 76-2039
TFM, 76-2039
Duck
DDT, 76-1768
Fish
Copper sulfate, 76-2008
Dichlorvos, 76-1973
Fenitrothion, 76-1973
Lindane, 76-1734
Naled, 76-1738
Phosmet, 76-1973
Temophos, 76-1991
Fungi
Herbicides, 76-2341
Gerbils
Mevinphos, 76-2318
Insects
Carbaryl, 76-2039
Dieldrin, 76-2039
Endothall, 76-2039
Malathion, 76-2039
Naled, 76-1738
TFM, 76-2039
Microorganisms
Atrazine, 76-2040
Carbaryl, 76-1822
Chlorazon, 76-2325
Chloroform, 76-2311
Cyanazine, 76-2040, 76-2041
Cycloate, 76-2325
2,4-D, 76-1990, 76-2040, 76-2041 76-2325
Dalapon, 76-2324
DNOC, 76-1822
Fungicides, 76-1822
MCPA, 76-1990
Molinate, 76-1990
Monuron, 76-2324
Phenmedipham, 76-2325
PMA, 76-2238
Pyrazon, 76-2325
Simazine, 76-2041, 76-2324
TCA, 76-2325
Temophos, 76-2012
Molluscs
DDT, 76-2056
Endosulfan, 76-2056
Mouse
Aziprotryn, 76-2343
Cystaphos, 76-1769
Toxicity/experimental animals (cont'd)
Paraquat, 76-1783
Plankton/algae
Chlorpyrifos, 76-1963
2,4-D, 76-1749, 76-1799
Diquat, 76-1799
Paraquat, 76-1799
Rotenone, 76-2322
Temophos, 76-1991, 76-2012
Plants
Herbicides, 76-2341
Rabbit
Aziprotryn, 76-2343
Phosmet, 76-2327
Rat
Azinphosmethyl, 76-1950
Aziprotryn, 76-2343
Benomyl, 76-2246
Dichlorvos, 76-2233
Dioxins, 76-1784
Fenitrothion, 76-2288
Fenitrothion derived compound 76-2288
Herbicides, 76-2321
Organochlorines, 76-2321
Paraquat, 76-1783
PCP, 76-1965
Validamycin, 76-2276
Reptiles
Naled, 76-1738
Toxicity/humans
General, 76-2198
Herbicides, 76-1936
Organochlorines, 76-1936
Organophosphates, 76-1936
Accidental, 76-1698
Arsenicals, 76-1678, 76-1685 76-2213
Blasticidin s, 76-2212
BPMC, 76-2212
Dichlorvos, 76-2212
Mercurials, 76-1680
Organophosphates, 76-1682 76-2221
Paraquat, 76-1933, 76-2212
Parathion, 76-1929
PCNB, 76-2212
Thallium, 76-1691, 76-2218
Trichlorfon, 76-1686, 76-1688
Zinc phosphide, 76-1676, 76-1677
Experimental
Pralidoxime, 76-1958
Intentional
Methyl demeton, 76-2208
Paraoxon, 76-2201
Paraquat, 76-1922, 76-1927
Parathion, 76-2201, 76-2222 76-2223
Thallium, 76-1697, 76-2210
Occupational, 76-1698, 76-1701 76-1702, 76-1703, 76-1934 76-2196, 76-2207
Aldicarb, 76-2203
Benomyl, 76-2203
Carbamates, 76-1687
Chlordane, 76-1935
Chlordecone, 76-1684, 76-1926
Toxicity/humans (cont'd)
DDT, 76-2203
Dichlorvos, 76-2203
Dioxacarb, 76-1689
Fenitrothion, 76-1704
Fungicides, 76-1705, 76-1706
Herbicides, 76-1705, 76-1706
Maneb, 76-1704
Mercurials, 76-1699
Organophosphates, 76-1687 76-1699, 76-1705, 76-1706 76-1924, 76-2199, 76-2206 76-2221, 76-2225
Paraquat, 76-1704
Pyrazon, 76-1923
2,4,5 T derived compound 76-1690
Thiophanate-methyl, 76-1707
Tin compounds, 76-1699
Zineb, 76-1930
Toxicity/non-target organisms
General
DDT, 76-1809, 76-1918
Organophosphates, 76-1918
Amphibians
DDT, 76-2200
Cat
Crimidine, 76-1695
Chicken
2,4,5 T derived compound 76-1690
Cow, 76-1953
2,4,5-T, 76-1953
Crustacea
Herbicides, 76-2220
Dog
Crimidine, 76-1695
Lead, 76-1696
Metaldehyde, 76-1696
Parathion, 76-1696
Strychnine, 76-1696
Thallium, 76-1696
Fish, 76-2226
Chlordecone, 76-1926
Endrin, 76-2224
Herbicides, 76-2220
Molluscs
Herbicides, 76-2220
Pheasant
Crimidine, 76-1695
Plants
2,4-D, 76-1953
2,4,5-T, 76-1953
Treatment of poisoning
General, 76-1681, 76-2198
DNOC, 76-1600
Nitrofen, 76-1600
Organochlorines, 76-2059
Organophosphates, 76-1600 76-2059, 76-2211
Paraquat, 76-1922
PCP, 76-1600
Rodenticides, 76-1679
Thallium, 76-1683, 76-1691
Atropine
Chlorothion, 76-1931

Treatment of poisoning (cont'd)
Dioxacarb, 76-1689
Malathion, 76-1931
Methyl demeton, 76-2208
Organophosphates, 76-1928
76-2199
Parathion, 76-1929
Trichlorfon, 76-1810
Bentonite
Paraquat, 76-1932
Berlin blue
Thallium, 76-1697
Fuller's earth
Paraquat, 76-1932
Hypoxia
Paraquat, 76-1982
Mannitol
Thallium, 76-1697
Oximes
DFP, 76-1971
Dichlorvos, 76-1971

Treatment of poisoning (cont'd)
Organophosphates, 76-1988
Pralidoxime, 76-1928
Organophosphates, 76-2199
Parathion, 76-1929
Propranolol
Thallium, 76-2210

Upper respiratory tract
see also Respiratory system
Animals/experimental
Diquat, 76-2277

Vision
see also Sensory system
Animals/experimental
Organochlorines, 76-1979
76-1981
Organophosphates, 76-2274
Trichlorophos, 76-1804
Human
Blasticidin s, 76-2212

Vision (cont'd)
BPMC, 76-2212
Dichlorvos, 76-2212
Dioxacarb, 76-1689
Mercurials, 76-2212
Paraquat, 76-1933, 76-2212
PCNB, 76-2212
Pralidoxime, 76-1958

Vitamins/coenzymes
see also Biochemical effects
Animals/experimental
DDT, 76-2013
Lindane, 76-2013
Animals/non-target
Ronnel, 76-1951

A-Rest
see Ancymidol

Abate
see Temophos

Abate 1-SG
see Temophos

Agent Orange
see 2,4-D; 2,4,5-T

Aldicarb
see also Carbamates
Peripheral nerves
Human, 76-2203
Residue degradation
Food and feed, 76-1882
Residues/food and feed
Vegetables, 76-1879, 76-1882
Toxicity/humans
Occupational, 76-2203

Aldrin
see also Organochlorines
Absorption
In vitro, 76-1962
Biotransformation
Annelids, 76-2287
Fish, 76-1985
Distribution/storage
Annelids, 76-2287
Enzyme activity
Mixed function oxidases, 76-1724 76-1985
Microsomes
In vitro, 76-1724
Residue degradation
Soil, 76-1617
Residues/water
Groundwater/rain, 76-2146

Allethrin
see also Botanicals; Pyrethrins
Chromatography
Gas-liquid, 76-2081

Altosid
see also Juvenile hormones and analogs
Amino acids/peptides/proteins
Animals/experimental, 76-1736
Nucleic acids
Animals/experimental, 76-1736

Altozar
see also Juvenile hormones and analogs
Amino acids/peptides/proteins
Animals/experimental, 76-1736
Nucleic acids
Animals/experimental, 76-1736

Amitrole
see also Herbicides
Biotransformation

Amitrole (cont'd)
Plants, 76-2004
Mutagenesis/teratogenesis
Animals/experimental, 76-2252
Microorganisms, 76-2252

Ancymidol
see also Herbicides
Analysis
Sample preparation, 76-2070 76-2070
Chromatography
Gas-liquid, 76-2070

Antibiotics
see Ezomycin A; Ezomycin B

Antidotes
Enzyme activity
Cholinesterase, 76-1988

Armine
see also Organophosphates
Enzyme activity
Cholinesterase, 76-2002
Muscle, striated
In vitro, 76-2002
Sympathetic nerves
In vitro, 76-2002

Arsenicals
Metabolism
Cow, 76-2054
Human, 76-2054
Morbidity and mortality statistics
Hungary, 76-1678
Nervous system
Human, 76-2213
Residues/food and feed
Dairy products, 76-2054
Fruits, 76-1862
Meat, 76-2054
Vegetables, 76-2054
Residues/humans
Urine, 76-2054
Residues/plants
Cotton, 76-2054
Residues/soil
Adsorption, 76-1660
Movement, 76-1660
Residues/water
Rivers/streams, 76-2136
Skin
Human, 76-1678
Spectrometry
Atomic absorption/emission 76-1824
Toxicity/humans
Accidental, 76-1678, 76-1685 76-2213

Arylam
see Carbaryl

Atrazine
see also Herbicides
Analysis

Atrazine (cont'd)
Sample preparation, 76-2094
Carcinogenesis
General, 76-1725
Chromatography
Gas-liquid, 76-2094
Enzyme activity
Invertase, 76-2029
Polygalacuronase, 76-2029
Factors influencing metabolism/toxicity
Schedule of dosage, 76-2040
Growth
Microorganisms, 76-2029
Nitrogen fixation
Microorganisms, 76-2294
Photodecomposition, 76-1725
Residue degradation
Plants, 76-1668
Soil, 76-1668, 76-2164
Water, 76-2149
Residues/soil
General, 76-1668
Adsorption, 76-2172
Residues/water
General, 76-1629
Spectrometry
Infrared, 76-1725
Mass spectrometry, 76-1725
NMR, 76-1725
UV, 76-1725
Toxicity/experimental animals
Chicken, 76-2017
Microorganisms, 76-2040

Azinphosmethyl
see also Organophosphates
Biotransformation
Plants, 76-2015
Enzyme activity
Cholinesterase, 76-1950
Factors influencing metabolism/toxicity
Sex, 76-1950
Growth
Animals/experimental, 76-1950
Toxicity/experimental animals
Rat, 76-1950

Aziprotryn
see also Herbicides
Toxicity/experimental animals
Mouse, 76-2343
Rabbit, 76-2343
Rat, 76-2343

BAY NTN 9306
see also Organophosphates
Biotransformation
Microorganisms, 76-2022
Plants, 76-2022

Bayluscide
see Clonitralide

Baythion
see Phoxim

Benefin
see also Herbicides
Electrometry
Polarography, 76-2064
Residue degradation
Soil, 76-2167

Benlate
see Benomyl

Benomyl
see also Fungicides
Analysis
Sample preparation, 76-1845
Behavior
Animals/experimental, 76-2266
Mitosis/meiosis
In vitro, 76-2042
Mutagenesis/teratogenesis
Animals/experimental, 76-2245
In vitro, 76-2245
Peripheral nerves
Human, 76-2203
Reproductive organs, male
Animals/experimental, 76-2246
Residues/plants
Orchard, 76-2134
Spectrometry
UV, 76-1845
Toxicity/experimental animals
Rat, 76-2246
Toxicity/humans
Occupational, 76-2203

Bentazon
see also Herbicides
Biotransformation
Plants, 76-1817, 76-2334, 76-2335
Distribution/storage
Plants, 76-1817
Factors influencing metabolism/toxicity
Age, 76-2335
Taxon, 76-2335

Betanal
see Phenmedipham

BHC
see also Organochlorines; Lindane
Amino acids/peptides/proteins
Animals/experimental, 76-1819
Biotransformation
Cow, 76-1782
Carbohydrates
Animals/experimental, 76-1819
Factors influencing metabolism/toxicity
Interactions, 76-2316
Taxon, 76-2316
Fibroblasts
Animals/experimental, 76-2314
Human, 76-2314
Growth
Microorganisms, 76-2299

BHC (cont'd)
Lipids/steroids/sterols
Animals/experimental, 76-1819
Liver
Animals/experimental, 76-1819
Placental transfer
Human, 76-1649
Residue degradation
Soil, 76-2178
Residues/food and feed
Total diet, 76-1614
Dairy products, 76-1610, 76-2135
Fish, 76-2160
Fruits, 76-1656
Meat, 76-1860
Residues/humans
General, 76-1648
Adipose, 76-1651
Blood, 76-1641, 76-1642, 76-1643 76-1649
Milk, 76-1647
Residues/non-target organisms
Fish, 76-1657
Residues/plants
Paper, 76-2152
Residues/soil
Movement, 76-2177, 76-2178
Residues/water
Groundwater/rain, 76-2146
Lakes/ponds, 76-1657
Reviews
Monitoring and residues, 76-1857

BHC isomers
Chromatography
Gas-liquid, 76-2071
Thin-layer, 76-2355
Enzyme activity
General, 76-1733
Factors influencing metabolism/toxicity
Interactions, 76-1733
Liver
Animals/experimental, 76-1823
Nucleic acids
Animals/experimental, 76-1823
Residues/food and feed
Total diet, 76-1874
Animal feed, 76-2154
Dairy products, 76-1653, 76-1915 76-2142
Residues/humans
Milk, 76-1874, 76-1913
Urine, 76-2174
Residues/non-target organisms
Fish, 76-1912

Bi-58
see Dimethoate

Birlane
see Chlorfenvinphos

Blasticidin S
see also Fungicides; Soil bactericide
Toxicity/humans
Accidental, 76-2212
Vision
Human, 76-2212

Botanicals
see Allethrin; Pyrethrin I; Rotenone

BPMC
see also Carbamates
Residue degradation
Soil, 76-2188
Toxicity/humans
Accidental, 76-2212
Vision
Human, 76-2212

BPMC derived compounds
Enzyme activity
Cholinesterase, 76-1730

Brevinyl E-50
see Dichlorvos

Burex
see Pyrazon

Butonate
see also Organophosphates
Nucleic acids
Animals/experimental, 76-2279

γ-BHC
see Lindane

Cadmium
see also Heavy metals
Residues/non-target organisms
Duck, 76-1634

Captan
see also Fungicides
Amino acids/peptides/proteins
Microorganisms, 76-2291
Enzyme activity
General, 76-1732
Gametogenesis
Microorganisms, 76-2291
Mitochondria
Plants, 76-1716
Mutagenesis/teratogenesis
Microorganisms, 76-2262
Nucleic acids
Microorganisms, 76-2291
Porphyrins
Plants, 76-1716
Residues/food and feed
Vegetables, 76-2186
Respiration, cellular
Plants, 76-1716

Carbamates
see also Aldicarb; BPMC; Carbaryl; Carbendazim; Carbofuran; Dioxacarb; Maneb; Nabam; Phenmedipham; Propoxur; Zineb
Analysis
Sample preparation, 76-1846
Biotransformation
Microorganisms, 76-2036
Plants, 76-2241
Carcinogenesis
General, 76-2049
Chromatography
Gas-liquid, 76-1826, 76-2078 76-2087

Carbamates (cont'd)
Thin-layer, 76-2065, 76-2082
Economics, 76-1598
EEG
Human, 76-1687
Environmental pollution, 76-2108
Metabolism
Rat, 76-2049
Mutagenesis/teratogenesis
General, 76-2049
Animals/experimental, 76-2305
Residue degradation
Soil, 76-2189
Skin
Animals/experimental, 76-2275
Toxicity/experimental animals
Birds, 76-1801
Toxicity/humans
Occupational, 76-1687

Carbaryl
see also Carbamates
Absorption
Chicken, 76-2320
Pig, 76-1812
Adrenal
Animals/experimental, 76-1790
Amino acids/peptides/proteins
Animals/experimental, 76-1763
Biotransformation
In vitro, 76-2283, 76-2284
Pig, 76-1812
Carcinogenesis
Animals/experimental, 76-2239 76-2240
Chromatography
Thin-layer, 76-1828
Cytological effects
Animals/experimental, 76-2251
Plants, 76-2251
Distribution/storage
Chicken, 76-2320
Pig, 76-1812
Enzyme activity
General, 76-1773
Cholinesterase, 76-1772
Esterases, 76-2283, 76-2284
GOT, 76-1772
GPT, 76-1772
Excretion
Sheep, 76-1811
Factors influencing metabolism/toxicity
Schedule of dosage, 76-2320
Fibroblasts
Animals/experimental, 76-2314
Human, 76-2314
Growth
Microorganisms, 76-1944, 76-1993 76-2342
Lipids/steroids/sterols
Animals/experimental, 76-1790 76-1791
Liver
Animals/experimental, 76-1763 76-1791
Metabolism
Rat, 76-1722

Carbaryl (cont'd)
Mutagenesis/teratogenesis
Microorganisms, 76-2267
Nucleic acids
Animals/experimental, 76-1763
Photodecomposition, 76-1891
Residue degradation
Soil, 76-1671
Water, 76-2149
Residues/food and feed
Dairy products, 76-1663
Fruits, 76-1609, 76-1914
Residues/soil
Adsorption, 76-2155
Movement, 76-1671, 76-2155
Respiration, cellular
In vitro, 76-2283, 76-2284
Spectrometry
Colorimetry, 76-1831, 76-1832
Thermoregulation
Animals/experimental, 76-1785 76-1785
Toxicity/experimental animals
Insects, 76-2039
Microorganisms, 76-1822

Carbaryl derived compound
Mutagenesis/teratogenesis
Microorganisms, 76-2016

Carbendazim
see also Carbamates; Fungicides
Behavior
Animals/experimental, 76-2266
Mitosis/meiosis
In vitro, 76-2042
Residues/plants
Orchard, 76-2134

Carbofuran
see also Carbamates
Biotransformation
Plants, 76-2038
Residue degradation
Soil, 76-2129
Residues/food and feed
Vegetables, 76-1896
Residues/soil
Movement, 76-2129

Carboxin
see also Fungicides
Biotransformation
Fungi, 76-1820

Ceresan M
see Ethyl mercury *p*-toluene sulfonamide

Chemosterilants
see also Tepa
Chromosomes/genes
Human, 76-1974
Factors influencing metabolism/toxicity
Adaptation, 76-1821
Mutagenesis/teratogenesis
Human, 76-1974

Chlorazon
see also Herbicides
Toxicity/experimental animals
Microorganisms, 76-2325

Chlordane
see also Organochlorines
Amino acids/peptides/proteins
Animals/experimental, 76-2280
Blood cells
Human, 76-1935
Carbohydrates
Animals/experimental, 76-2235 76-2280
Enzyme activity
Acid phosphatase, 76-2280
Alkaline phosphatase, 76-2280
Factors influencing metabolism/toxicity
Electroshock, 76-1774
Interactions, 76-1774
Metabolism
Rat, 76-1722
Pancreas (endocrine)
Animals/experimental, 76-2235
Residues/food and feed
Vegetables, 76-1881
Residues/water
Rivers/streams, 76-2140
Toxicity/humans
Occupational, 76-1935

Chlordecone
see also Organochlorines
Lipids/steroids/sterols
Human, 76-2045
Nervous system
Human, 76-1684, 76-1926 76-2045
Toxicity/humans
Occupational, 76-1684, 76-1926
Toxicity/non-target organisms
Fish, 76-1926

Chlordimeform
see also Organochlorines
Absorption
In vitro, 76-1962

Chlorfenvinphos
see also Organophosphates
Biochemical effects
General, 76-2248
Digestive glands
Animals/experimental, 76-2248
Enzyme activity
Cholinesterase, 76-2248

Chlorfenvinphos derived compound
Amino acids/peptides/proteins
In vitro, 76-1727
Biotransformation
In vitro, 76-1727

2-Chlorobenzamide
see also Herbicides
Respiration, cellular
Microorganisms, 76-1742

Chloroform
see also Fumigants
Experimental design
Toxicology and pharmacology
76-2313
Factors influencing metabolism/toxicity
Nutritional state, 76-2310
pH, 76-2310
Growth
Microorganisms, 76-2311, 76-2312
Nitrogen fixation
Microorganisms, 76-2309, 76-2310 76-2311, 76-2312
Respiration, cellular
Microorganisms, 76-2309, 76-2312
Toxicity/experimental animals
Microorganisms, 76-2311

Chloronaphthalenes
see also Fungicides
Biotransformation
Pig, 76-2018

Chlorophos
see Trichlorfon

Chlorothalonil
see also Fungicides
Residues/food and feed
Fruits, 76-2192

Chlorothion
see also Organophosphates
Enzyme activity
Cholinesterase, 76-1931
Treatment of poisoning
Atropine, 76-1931

Chloroxuron
see also Herbicides
Carcinogenesis
Microorganisms, 76-2256
Metabolism
Microorganisms, 76-2256

Chlorpropham
see also Herbicides
Biotransformation
Plants, 76-2020

Chlorpyrifos
see also Organophosphates
Biotransformation
Plankton/algae, 76-2237
Growth
Microorganisms, 76-2237
Toxicity/experimental animals
Plankton/algae, 76-1963

Chlorpyrifos-methyl
see also Organophosphates
Metabolism
Sheep, 76-2034

Chlortoluron
see also Herbicides
Biotransformation
In vitro, 76-2283, 76-2284

Chlortoluron (cont'd)
Enzyme activity
Esterases, 76-2283, 76-2284
Respiration, cellular
In vitro, 76-2283, 76-2284

CIPC
see Chloropropham

CKB 1028
see also Organophosphates
Enzyme activity
Cholinesterase, 76-1940
Esterases, 76-1940

Clonitralide
see also Molluscicide
Residue degradation
Soil, 76-1613

CNP
see also Herbicides
Residue degradation
Soil, 76-2183

Co-Ral
see Coumaphos

Copper
Spectrometry
Colorimetry, 76-2353

Copper sulfate
see also Fungicides
Liver
Animals/experimental, 76-2008
Toxicity/experimental animals
Fish, 76-2008

Coumaphos
see also Organophosphates
Adrenal
Animals/experimental, 76-1790
Lipids/steroids/sterols
Animals/experimental, 76-1790 76-1791
Liver
Animals/experimental, 76-1791

Counter
see Terbufos

Crimidine
see also Juvenile hormones and analogs; Rodenticides
Toxicity/non-target organisms
Cat, 76-1695
Dog, 76-1695
Pheasant, 76-1695

Cyanazine
see also Herbicides
Biotransformation
Plants, 76-2336
Factors influencing metabolism/toxicity
Schedule of dosage, 76-2040
Growth
Microorganisms, 76-2041
Toxicity/experimental animals
Microorganisms, 76-2040, 76-2041

Cyanophos
see also Organophosphates
Residues/food and feed
Vegetables, 76-2179

Cyanox
see Cyanophos

Cycloate
see also Herbicides
Toxicity/experimental animals
Microorganisms, 76-2325

Cycluron
see also Herbicides
Metabolism
Microorganisms, 76-2256

Cyperquat
see also Herbicides
Chromatography
Gas-liquid, 76-2093

Cystaphos
see also Organophosphates
Factors influencing metabolism/toxicity
Route, 76-1769
Toxicity/experimental animals
Mouse, 76-1769

2,4-D
see also Herbicides
Absorption
Plankton/algae, 76-1748
Biotransformation
Plants, 76-1943
Cell membranes
Microorganisms, 76-1748
Chromatography
Gas-liquid, 76-2088, 76-2091
Chromosomes/genes
In vitro, 76-1786
Factors influencing metabolism/toxicity
Adaptation, 76-1990
Schedule of dosage, 76-2040
Taxon, 76-1749, 76-1990
Growth
Microorganisms, 76-2041
Muscle, striated
Animals/experimental, 76-1739
Mutagenesis/teratogenesis
Animals/non-target, 76-1953
Nitrogen cycle
Microorganisms, 76-1956
Photodecomposition, 76-1902
Residue degradation
Soil, 76-1672
Water, 76-2149
Residues/soil
Volatilization, 76-2165
Residues/water
Groundwater/rain, 76-1906
Irrigation, 76-1655
Rivers/streams, 76-2125
Toxicity/experimental animals
Microorganisms, 76-1990, 76-2040 76-2041, 76-2325
Plankton/algae, 76-1749, 76-1799

2,4-D (cont'd)
Toxicity/non-target organisms
Plants, 76-1953

Dalapon
see also Herbicides
Toxicity/experimental animals
Microorganisms, 76-2324

Daminozide
see also Herbicides
Analysis
Sample preparation, 76-2070
Chromatography
Gas-liquid, 76-2070

Dazomet
see also Nematocides
Respiration, cellular
Microorganisms, 76-1742

2,4-DB
see also Herbicides
Mitochondria
Plants, 76-1716
Porphyrins
Plants, 76-1716
Respiration, cellular
Plants, 76-1716

DCMO
see Carboxin

DCNA
see Dichloran

DDD
see TDE

DDE
see also Organochlorines
Analysis
Sample preparation, 76-2094
Chromatography
Gas-liquid, 76-2094
Chromosomes/genes
Animals/experimental, 76-2263
Distribution/storage
Human, 76-1967
Factors influencing metabolism/toxicity
Structure/function, 76-1948
Growth
Microorganisms, 76-2299
Lipids/steroids/sterols
Human, 76-1967
Mutagenesis/teratogenesis
Animals/experimental, 76-2263
Plasma/serum
Human, 76-1967
Residues/food and feed
Animal feed, 76-1865, 76-2154
Dairy products, 76-1653, 76-1876 76-2142
Fruits, 76-1656
Poultry, 76-1876
Residues/humans
Adipose, 76-1651
Milk, 76-1647, 76-1651
Residues/non-target organisms
Duck, 76-1637

DDE (cont'd)
Residues/water
General, 76-1629
Groundwater/rain, 76-2146
Oceans/seas, 76-1877

DDT
see also Organochlorines
Absorption
In vitro, 76-1962
Bioassay, 76-2356
Biochemical effects
General, 76-1760, 76-2048
Biotransformation
General, 76-1760
Chicken, 76-1782
Cow, 76-1782
Blood cells
In vitro, 76-2264
Carbohydrates
Human, 76-1753
Carcinogenesis
General, 76-1675
Chromatography
Gas-liquid, 76-2071, 76-2100 76-2344
Thin-layer, 76-2355
Chromosomes/genes
Animals/experimental, 76-2263
In vitro, 76-2264
Distribution/storage
General, 76-1760
Fish, 76-2330
Embryo/fetus
Animals/experimental, 76-1969
Endocrine system
Animals/experimental, 76-2013
Environmental pollution, 76-1601 76-1606
Enzyme activity
General, 76-1718, 76-1733
Mixed function oxidases, 76-1753 76-1760, 76-2317
Factors influencing metabolism/toxicity
Age, 76-2330
Biological magnification, 76-1809
Formulation, 76-2304
Interactions, 76-1718, 76-1733 76-1903, 76-2304
Nutritional state, 76-2048 76-2317
Taxon, 76-1718, 76-1969
Fibroblasts
Animals/experimental, 76-2314
Human, 76-2314
Gonads
Animals/experimental, 76-1969
Growth
Microorganisms, 76-1944, 76-1993 76-2299
Immunology
In vitro, 76-2001
Kidney
Animals/experimental, 76-1939
Laws and regulations
Spain, 76-1918
USA, 76-1675

DDT (cont'd)
Liver
Animals/experimental, 76-1760 76-1782, 76-1939, 76-1983
Human, 76-1760
Metabolism
Amphibians, 76-1960
Hamster, 76-2337
Mouse, 76-1741, 76-2337
Muscle, striated
Animals/experimental, 76-2296
Mutagenesis/teratogenesis
Animals/experimental, 76-2263
In vitro, 76-2264
Peripheral nerves
Human, 76-2203
Photodecomposition, 76-1884
Residue degradation
Soil, 76-1617, 76-2129, 76-2178
Water, 76-2159
Residue removal
Food and feed, 76-1901, 76-2161
Water, 76-2122
Residues/food and feed
General, 76-1885
Total diet, 76-1614
Animal feed, 76-1865, 76-1903 76-2154
Baby formula, 76-2120
Dairy products, 76-1653, 76-1663 76-1915, 76-2135, 76-2142
Fish, 76-2160
Fruits, 76-1656, 76-2175
Meat, 76-1860
Vegetables, 76-2175
Residues/humans
General, 76-1648
Adipose, 76-1651
Blood, 76-1641, 76-1642, 76-1643
Milk, 76-1647, 76-1651, 76-1861 76-1913
Residues/non-target organisms
General, 76-1809
Crustacea, 76-2145
Duck, 76-1634, 76-1637
Fish, 76-1636, 76-1657, 76-1912 76-2139
Microorganisms, 76-2137
Plankton/algae, 76-2145
Seal, 76-2150
Residues/soil
Adsorption, 76-2137, 76-2138
Movement, 76-2129, 76-2138 76-2177, 76-2178
Volatilization, 76-1619
Residues/water
Groundwater/rain, 76-2146
Lakes/ponds, 76-1657
Oceans/seas, 76-1877, 76-2139
Rivers/streams, 76-2139, 76-2140
Respiration, cellular
Animals/experimental, 76-1983 76-2056
Reviews
Monitoring and residues, 76-1857
Thermoregulation
Animals/experimental, 76-1785

DDT (cont'd)
Animals/experimental, 76-1785
Toxicity/experimental animals
Duck, 76-1768
Molluscs, 76-2056
Toxicity/humans
Occupational, 76-2203
Toxicity/non-target organisms
General, 76-1809, 76-1918
Amphibians, 76-2200
Vitamins/coenzymes
Animals/experimental, 76-2013

DDT derived compounds
Amino acids/peptides/proteins
In vitro, 76-2285
Placental transfer
Human, 76-1649
Plasma/serum
In vitro, 76-2285
Residues/humans
Blood, 76-1649
Spectrometry
Colorimetry, 76-1825
UV, 76-1825

DDT isomers
Amino acids/peptides/proteins
Animals/experimental, 76-1996
Biotransformation
Rat, 76-2000
Brain
Animals/experimental, 76-1996
Distribution/storage
Human, 76-1967
Eggshell effects
Animals/experimental, 76-1721
Embryo/fetus
Animals/experimental, 76-1969
Excretion
Rat, 76-2000
Factors influencing metabolism/toxicity
Structure/function, 76-1948
Taxon, 76-1969
Gonads
Animals/experimental, 76-1969
Kidney
Animals/experimental, 76-1996
Lipids/steroids/sterols
Human, 76-1967
Plasma/serum
Human, 76-1967
Residues/food and feed
Total diet, 76-1874
Dairy products, 76-1876
Poultry, 76-1876
Residues/humans
Adipose, 76-1870
Milk, 76-1874

Decemtion P-60
see Phosmet

Demephion
see also Organophosphates
Enzyme activity
Cholinesterase, 76-1940
Esterases, 76-1940

Desmetryne
see also Herbicides
Residue degradation
Water, 76-2149

DFP
see also Organophosphates
Biotransformation
Squid, 76-2043
Enzyme activity
Cholinesterase, 76-1942
Oximes, 76-1971
Factors influencing metabolism/toxicity
Taxon, 76-1971
Peripheral nerves
Animals/experimental, 76-2043
Treatment of poisoning
Oximes, 76-1971

Diazinon
see also Organophosphates
Analysis
Sample preparation, 76-2062
Chromatography
Gas-liquid, 76-2062
Mutagenesis/teratogenesis
Microorganisms, 76-2016
Residues/food and feed
General, 76-2131
Fruits, 76-1627
Vegetables, 76-1627

Dibrom
see Naled

1,2- Dibromoethane
see also Fumigants
Residues/soil
Movement, 76-1887

Dicamba
see also Herbicides
Biotransformation
Plants, 76-2014
Residue degradation
Plants, 76-1880
Soil, 76-1880
Residues/plants
Forest, 76-2141
Residues/soil
Movement, 76-2141

Dichloran
see also Fungicides
Biotransformation
Microorganisms, 76-2019

1,3-Dichloropropene
see Telone; Fumigants

Dichlorvos
see also Organophosphates
Adrenal
Animals/experimental, 76-1968
Amino acids/peptides/proteins
In vitro, 76-2058
Analysis
Sample preparation, 76-2063

Dichlorvos (cont'd)
Brain
Animals/experimental, 76-2269 76-2271
Enzyme activity
Acid phosphatase, 76-2271
Aliesterase, 76-1971
Alkaline phosphatase, 76-2271
Cholinesterase, 76-1971, 76-2271
Cytochrome oxidase, 76-2269 76-2272
Succinic dehydrogenase, 76-2269 76-2272
Factors influencing metabolism/toxicity
Taxon, 76-1971, 76-1971
Fertility/sterility
Animals/experimental, 76-1964
Fibroblasts
In vitro, 76-2058
Growth
Microorganisms, 76-2342
Metabolism
Cow, 76-1797
Mitosis/meiosis
Microorganisms, 76-2258
Mutagenesis/teratogenesis
Animals/experimental, 76-2261
Microorganisms, 76-2249
Peripheral nerves
Human, 76-2203
Residues/water
Rivers/streams, 76-1628
Skin
Animals/experimental, 76-1959 76-2233
Spectrometry
Colorimetry, 76-1852
Therapeutic use
Demodicidosis, 76-1959
Toxicity/experimental animals
Fish, 76-1973
Rat, 76-2233
Toxicity/humans
Accidental, 76-2212
Occupational, 76-2203
Treatment of poisoning
Oximes, 76-1971
Vision
Human, 76-2212

Dicofol
see also Organochlorines
Biotransformation
Insects, 76-1720

Dicumarol
see also Rodenticides
Spectrometry
UV, 76-2076

Dieldrin
see also Organochlorines
Absorption
In vitro, 76-1962
Amino acids/peptides/proteins
Microorganisms, 76-2291
Analysis
Sample preparation, 76-2094

Dieldrin (cont'd)
Behavior
Animals/experimental, 76-2050
Biochemical effects
General, 76-2048
Biotransformation
Microorganisms, 76-2055
Carcinogenesis
Animals/experimental, 76-2303
Chromatography
Gas-liquid, 76-2071, 76-2094
EEG
Animals/experimental, 76-1779
Enzyme activity
Mixed function oxidases, 76-1724
Factors influencing metabolism/toxicity
Age, 76-2303
Interactions, 76-2053
Nutritional state, 76-2048
Hormones
Animals/experimental, 76-2053
Liver
Animals/experimental, 76-1782 76-2053, 76-2303
Microsomes
In vitro, 76-1724
Nucleic acids
Microorganisms, 76-2291
Reproduction/growth
Animals/experimental, 76-1802 76-1997
Residue degradation
Soil, 76-1617
Residue removal
Water, 76-2157
Residues/food and feed
Total diet, 76-1614
Animal feed, 76-1865, 76-2154
Dairy products, 76-1610
Residues/humans
General, 76-1648
Residues/non-target organisms
Crustacea, 76-1638
Duck, 76-1634, 76-1637
Fish, 76-1636, 76-1638
Insects, 76-1638
Seal, 76-2150
Residues/soil
Adsorption, 76-2138
Movement, 76-2138
Volatilization, 76-1899
Residues/water
General, 76-1629
Groundwater/rain, 76-2146
Rivers/streams, 76-1638
Toxicity/experimental animals
Crustacea, 76-2039
Insects, 76-2039

Diethoxyphosphorylothiocholine iodide
see Phosoline

Difluron
see also Fluorine compounds
Metabolism
Cow, 76-2028

Dimefox
see also Organophosphates
Carcinogenesis
Microorganisms, 76-2256
Metabolism
Microorganisms, 76-2256

Dimethoate
see also Organophosphates
Analysis
Sample preparation, 76-2101
Chromatography
Column, 76-2101
Thin-layer, 76-2101, 76-2350
Enzyme activity
Cholinesterase, 76-1940
Esterases, 76-1940
Growth
Microorganisms, 76-1944
Residue degradation
Plants, 76-1888
Residues/food and feed
Vegetables, 76-1888
Residues/plants
Medicinals and condiments 76-1888
Toxicity/experimental animals
Bee, 76-1755

Dinitramine
see also Herbicides
Electrometry
Polarography, 76-2064

Dinoseb
see also Herbicides
Biotransformation
Rat, 76-1946
Liver
Animals/experimental, 76-1946
Porphyrins
Animals/experimental, 76-1946

Dioxacarb
see also Carbamates
Toxicity/humans
Occupational, 76-1689
Treatment of poisoning
Atropine, 76-1689
Vision
Human, 76-1689

Dioxins
see also Polychlorinated biphenyls; 2,4,5-T
Biochemical effects
General, 76-1784
Liver
Animals/experimental, 76-1784
Thymus
Animals/experimental, 76-1784
Toxicity/experimental animals
Rat, 76-1784

Dipterex
see Trichlorfon

Diquat
see also Herbicides
Growth
Microorganisms, 76-2265

Diquat (cont'd)
Nitrogen fixation
Microorganisms, 76-2265
Toxicity/experimental animals
Plankton/algae, 76-1799
Upper respiratory tract
Animals/experimental, 76-2277

Disulfoton
see also Organophosphates
Nitrogen fixation
Microorganisms, 76-2292
Residue degradation
Soil, 76-1908
Residues/food and feed
Vegetables, 76-1879

Disyston
see Disulfoton

Diuron
see also Herbicides
Residue degradation
Soil, 76-2164

DNBP
see Dinoseb

DNOC
see also Fungicides
Toxicity/experimental animals
Microorganisms, 76-1822
Treatment of poisoning
General, 76-1600

Dodine
see also Fungicides
Mutagenesis/teratogenesis
Microorganisms, 76-2267

Dursban
see Chlorpyrifos

EL-273
see Triarimol

Endosulfan
see also Organochlorines
Biotransformation
Microorganisms, 76-2055
Plants, 76-2023
Residue degradation
Soil, 76-1617
Residues/plants
Medicinals and condiments 76-1867
Respiration, cellular
Animals/experimental, 76-2056
Toxicity/experimental animals
Molluscs, 76-2056

Endothall
see also Herbicides
Toxicity/experimental animals
Crustacea, 76-2039
Insects, 76-2039

Endrin
see also Organochlorines
Residue removal
Water, 76-2122
Residues/food and feed
Animal feed, 76-2154

Endrin (cont'd)
Reviews
Toxicology and pharmacology 76-2231
Toxicity/non-target organisms
Fish, 76-2224

Eptam
see EPTC

Ethephon
see also Organophosphates
Biotransformation
Plants, 76-1717

Ethion
see also Organophosphates
Biotransformation
Plants, 76-2023
Distribution/storage
Turkey, 76-2031
Residues/food and feed
Vegetables, 76-1896

Ethirimol
see Fungicides

Ethylene dibromide
see also Fumigants
Amino acids/peptides/proteins
Animals/experimental, 76-2047
Gametogenesis
Animals/experimental, 76-2047

Ethylene dichloride
see also Fumigants
Chromatography
Gas-liquid, 76-2351

Ethylene thiourea
see also Fungicides
Biotransformation
Plants, 76-2021
Chromatography
Gas-liquid, 76-2347
Thin-layer, 76-2347
Factors influencing metabolism/toxicity
Schedule of dosage, 76-2306
Mutagenesis/teratogenesis
Animals/experimental, 76-2250 76-2253, 76-2306, 76-2307
Microorganisms, 76-2250

Ethylmercury chloride
see also Fungicides
Chromosomes/genes
Plants, 76-1787

Ethylmercury *p*-toluene sulfonamide
see also Fungicides
Mutagenesis/teratogenesis
Animals/experimental, 76-2260

Ezomycin A
see also Antibiotics
Bioassay, 76-1837

Ezomycin B
see also Antibiotics
Bioassay, 76-1837

Famophos
see Famphur

Famphur
see also Organophosphates
Biochemical effects
General, 76-1771
Blood cells
Animals/experimental, 76-1771
Chromatography
Thin-layer, 76-2099
Enzyme activity
Cholinesterase, 76-1771

FCPA
see Potassium-2-formyl- 1,4-chlorophenoxyacetate

Fenaminosulf
see also Fungicides
Carbohydrates
In vitro, 76-1938
Enzyme activity
NADH dehydrogenase, 76-1938

Fenchlorphos
see Ronnel

Fenitrothion
see also Organophosphates
Absorption
Plants, 76-1970
Analysis
Sample preparation, 76-2062
Biotransformation
Plants, 76-1970
Chromatography
Gas-liquid, 76-2062
Distribution/storage
Monkey, 76-1650
Embryo/fetus
Plants, 76-1970
Factors influencing metabolism/toxicity
Formulation, 76-2304
Interactions, 76-2304, 76-2316
Taxon, 76-2316
Nitrogen fixation
Microorganisms, 76-2308
Residue degradation
Soil, 76-2129
Residue dynamics, 76-2123
Residues/food and feed
Fruits, 76-2123, 76-2182
Residues/non-target organisms
Monkey, 76-1650
Plants, 76-2123
Residues/soil
Movement, 76-2129
Toxicity/experimental animals
Bee, 76-2297
Fish, 76-1973
Rat, 76-2288

Fenitrothion (cont'd)
Toxicity/humans
Occupational, 76-1704

Fenitrothion derived compound
Enzyme activity
Cholinesterase, 76-2288
Metabolism
In vitro, 76-2288
Mutagenesis/teratogenesis
Animals/experimental, 76-2288
Toxicity/experimental animals
Rat, 76-2288

Fenoprop
see Silvex

Fensulfothion
see also Organophosphates
Residue degradation
Soil, 76-2129
Residues/food and feed
Vegetables, 76-1896
Residues/soil
Movement, 76-2129

Fenthion
Toxicity/experimental animals
Bee, 76-2297

Fentin acetate
see also Fungicides; Molluscicide
Spectrometry
Colorimetry, 76-1843

Ferbam
see also Fungicides
Metabolism
Sheep, 76-2026

Fluorine compounds
see Difluron

Folithion
see Fenitrothion

Foschlor
see Trichlorfon

Fumigants
see also Chloroform; 1,2-dibromoethane; 1,-3-Dichloropropene; Ethylene dibromide; Ethylene dichloride; Hexachlorobutadiene; Methyl bromide
Enzyme activity
General, 76-1808, 76-1808
Mutagenesis/teratogenesis
Microorganisms, 76-1808
Respiration, cellular
Microorganisms, 76-1808

Fundazol
see Benomyl

Fungicides
see also Ethyl mercury *p*-toluene sulfonamide; Benomyl; Blasticidin S; Captan; Carbendazim; Carboxin; Chloronaphthalenes; Chlorthalonil; Copper sulfate;

Fungicides (cont'd)
Dichloran; DNOC; Dodine; Ethirimol; Ethylene thiourea; Ethylmercuric chloride; Fenaminosulf; Fentin acetate; Ferbam; Hexachlorobenzene; Hexachlorophene; Hymexazol; Inezin; Maneb; Methylmercury dicyandiamide; Monox; Nabam; Oxythioquinox; PCNB; PMA; Thiabendazole; Thiophanate; Thiophanate-methyl; Thiram; Triarimol; Triforine; Validamycin; Zineb
Analysis
General, 76-2068
Biotransformation
Fungi, 76-1977
Microorganisms, 76-2036
Carcinogenesis
General, 76-2049
Chromatography
Gas-liquid, 76-2087
Thin-layer, 76-2065
Chromosomes/genes
Human, 76-1974
Environmental pollution, 76-1854 76-2108
Immunology
In vitro, 76-2001
Metabolism
Rat, 76-2049
Mitosis/meiosis
Microorganisms, 76-2255
Mutagenesis/teratogenesis
General, 76-2049
Animals/experimental, 76-2305
Human, 76-1974
Microorganisms, 76-1808, 76-2254 76-2257
Residue degradation
Soil, 76-1644
Residues/air
Industrial, 76-2173
Residues/food and feed
Fruits, 76-1662
Residues/humans
Urine, 76-2173
Residues/soil
General, 76-1644
Respiration, cellular
Microorganisms, 76-1808
Reviews
Monitoring and residues, 76-2107
Skin
Animals/experimental, 76-2275
Toxicity/experimental animals
Birds, 76-1801
Microorganisms, 76-1822
Toxicity/humans
Occupational, 76-1705, 76-1706

Gardona
see Tetrachlorvinphos

Glyphosate
see also Herbicides
Biotransformation
Microorganisms, 76-1816
Chromatography
Gas-liquid, 76-2080
Residue degradation
Soil, 76-2168
Residues/soil
Adsorption, 76-1816, 76-2168
Movement, 76-1816, 76-2168
Spectrometry
Mass spectrometry, 76-2080

Gramoxone
see Paraquat

Granosan
see Ethyl mercuric chloride
Distribution/storage
Chicken, 76-2328
Pig, 76-1806
Enzyme activity
General, 76-1806
Kidney
Animals/non-target, 76-1806
Liver
Animals/non-target, 76-1806
Residue removal
Food and feed, 76-2161

GS-14254
see Secbumeton

HCH
see BHC

Heavy metals
see also Cadmium; Lead
Growth
Microorganisms, 76-2299

HEOD
see Dieldrin

HEOM
Biotransformation
In vitro, 76-2282
Insects, 76-2282

Heptachlor
see also Organochlorines
Adrenal
Animals/experimental, 76-1951
Catecholamines
Animals/non-target, 76-1951 76-1951
Chromatography
Gas-liquid, 76-2355
Enzyme activity
Cholinesterase, 76-1951
Growth
Animals/experimental, 76-1951
Microorganisms, 76-2299
Liver
Animals/experimental, 76-1939 76-1951
Residue removal
Water, 76-2122
Residues/soil
Adsorption, 76-2138

Heptachlor (cont'd)
Movement, 76-2138
Volatilization, 76-1899

Heptachlor epoxide
see also Organochlorines
Residue removal
Water, 76-2157

Herbicides
see also Chlorpropham, Amitrole; Ancymidol; Atrazine; Aziprotryn; Benefin; Bentazon; Chlorazine; Chlorazon; 2-Chlorobenzamide Chloroxuron; Chlortoluron; CNP; Cyanazine; Cycloate; Cycluron; Cyperquat; 2,4-D; Dalapon; Daminozide; 2,4-DB; Desmetryne; Dicamba; Dinitramine; Dinoseb; Diquat; Diuron Endothall; Glyphosate; Hexaflurate; Isopropalin; Linuron; Maleic hydrazide; MCPA; Methabenzthiazuron; Metribuzin; Molinate; Monuron; MSMA; Naphthaleneacetic acid; Nitralin; Nitrofen; Oryzalin; Oxadiazon; Paraquat; PCP; Perfluidone; Phenmedipham; Picloram; PMA; Prometryne; Pronamide; Propanil; Propham Protect; Pyrazon; Schradan; Secbumeton; Silvex; Simazine; 2,4,5-T; TCA; Tebuthiuron; Terbutryne; Trifluralin
Analysis
General, 76-2069
Sample preparation, 76-2070
Behavior
Animals/experimental, 76-2232
Beneficial effects, 76-2113
Biochemical effects
Microorganisms, 76-2326
Biotransformation
Microorganisms, 76-2064
Plants, 76-1715
Chromatography
Gas-liquid, 76-1826, 76-2070 76-2078, 76-2085
Chromosomes/genes
Human, 76-1974
Environmental pollution, 76-2113
Enzyme activity
General, 76-1808
Factors influencing metabolism/toxicity
Structure/function, 76-2321
Taxon, 76-1715, 76-2326
Growth
Microorganisms, 76-2341
Mitosis/meiosis
Microorganisms, 76-2257
Mutagenesis/teratogenesis
Human, 76-1974
Microorganisms, 76-1808, 76-2254 76-2257

Herbicides (cont'd)
Photodecomposition, 76-1866
Residue degradation
Soil, 76-1644, 76-2132
Residue dynamics, 76-1622
Residues/food and feed
Cereals, 76-1658
Vegetables, 76-1658
Residues/soil
General, 76-1644, 76-1659
76-1866
Adsorption, 76-2153
Residues/water
Groundwater/rain, 76-2153
Respiration, cellular
Microorganisms, 76-1808
Reviews
Monitoring and residues, 76-2107
Toxicity/experimental animals
Bee, 76-1756
Birds, 76-1801
Fungi, 76-2341
Plants, 76-2341
Rat, 76-2321
Toxicity/humans
General, 76-1936
Occupational, 76-1705, 76-1706
Toxicity/non-target organisms
Crustacea, 76-2220
Fish, 76-2220
Molluscs, 76-2220

Hexachlorobenzene
see also Fungicides
Chromatography
Gas-liquid, 76-2071
Distribution/storage
Mouse, 76-1776
Rat, 76-1776, 76-1999
Enzyme activity
Esterases, 76-1781
Factors influencing metabolism/
toxicity
Interactions, 76-1781
Growth
Microorganisms, 76-2299
Kidney
Animals/experimental, 76-1939
Liver
Animals/experimental, 76-1939
76-1999
Mutagenesis/teratogenesis
Animals/experimental, 76-1776
Porphyrins
Animals/experimental, 76-1999
Residues/food and feed
General, 76-2151
Animal feed, 76-1876, 76-1894
Dairy products, 76-1876
Poultry, 76-1876
Residues/humans
Adipose, 76-2151
Residues/non-target organisms
Crustacea, 76-2116
Duck, 76-1634
Fish, 76-2116
Residues/water
General, 76-2151

Hexachlorobenzene (cont'd)
Rivers/streams, 76-2116

Hexachlorobutadiene
see also Fumigants
Residues/non-target organisms
Crustacea, 76-2116
Fish, 76-2116
Residues/water
Rivers/streams, 76-2116

Hexachlorophene
see also Fungicides
Mutagenesis/teratogenesis
Animals/experimental, 76-1751

Hexaflurate
see also Herbicides
Analysis
Sample preparation, 76-1851

Hymexazol
see also Fungicides
Chromatography
Gas-liquid, 76-2349

IDIMP
see also Organophosphates
Biotransformation
In vitro, 76-1972
Enzyme activity
Cholinesterase, 76-1972

Imidan
see Phosmet

Inezin
see also Fungicides; Organophosphates
Photodecomposition, 76-2126

Isopropalin
see also Herbicides
Biotransformation
Microorganisms, 76-2333
Plants, 76-1815, 76-2333
Electrometry
Polarography, 76-2064

Juvenile hormones and analogs
see also Altosid; Altozar; Crimidine; Methoprene; Ro 10-3108; Strychnine
Analysis
General, 76-2066
Factors influencing metabolism/
toxicity
Adaptation, 76-1821

Kepone
see Chlordecone

Lead
see also Heavy metals
Residues/food and feed
Total diet, 76-1614
Fruits, 76-2191
Vegetables, 76-2191
Residues/non-target organisms
Duck, 76-1634
Residues/water
Rivers/streams, 76-2136
Toxicity/non-target organisms

Lead (cont'd)
Dog, 76-1696

Lebaycid
see Fenthion

Leptophos
see also Organophosphates
Biotransformation
Insects, 76-2278, 76-2278
Enzyme activity
Cholinesterase, 76-1777
Metabolism
Rat, 76-1722
Nervous system
Animals/experimental, 76-1777
Spinal cord
Animals/experimental, 76-2044

Lindane
see also BHC, Organochlorines
Adrenal
Animals/experimental, 76-1968
Biotransformation
Microorganisms, 76-2055
Plants, 76-1978, 76-2035
Rat, 76-1729, 76-2247
Blood cells
Animals/experimental, 76-2293
Distribution/storage
Pheasant, 76-2037
Sheep, 76-1796
EEG
Animals/experimental, 76-2244
Embryo/fetus
Animals/experimental, 76-1949
76-2037
Endocrine system
Animals/experimental, 76-2013
Enzyme activity
Mixed function oxidases, 76-1729
Factors influencing metabolism/
toxicity
Interactions, 76-1729
Season, 76-2035
Temperature, 76-1734
Fertility/sterility
Animals/experimental, 76-1949
Growth
Animals/experimental, 76-1949
Mitosis/meiosis
In vitro, 76-2042
Prevention
Decontamination, 76-1937
Residue degradation
Water, 76-2124
Residue removal
Food and feed, 76-2161
Water, 76-2157
Residues/food and feed
Animal feed, 76-1865
Dairy products, 76-1663
Meat, 76-1860
Residues/soil
Volatilization, 76-2124
Residues/water
Groundwater/rain, 76-2146
Rivers/streams, 76-2140
Toxicity/experimental animals

Lindane (cont'd)
Fish, 76-1734
Vitamins/coenzymes
Animals/experimental, 76-2013

Linuron
see also Herbicides
Biotransformation
Microorganisms, 76-1976
Mutagenesis/teratogenesis
Microorganisms, 76-2016
Residue degradation
Soil, 76-2164

Malathion
see also Organophosphates
Biotransformation
Plants, 76-2032
Rat, 76-1710
Carbohydrates
Animals/experimental, 76-1710 76-2331
Cell membranes
Animals/experimental, 76-1710
Chromatography
Gas-liquid, 76-2090
Gel permeation, 76-1830
Enzyme activity
Alkaline phosphatase, 76-1994
Cholinesterase, 76-1931
Esterases, 76-1775, 76-1781 76-1955
6-GPD, 76-2331
Lactic dehydrogenase, 76-2331
Succinic dehydrogenase, 76-1994
Factors influencing metabolism/ toxicity
Humidity, 76-2032
Interactions, 76-1781
Light/radiation, 76-1994
Fertility/sterility
Animals/experimental, 76-1964
Growth
Microorganisms, 76-1944
Nitrogen fixation
Microorganisms, 76-2308
Residue degradation
Plants, 76-1888
Water, 76-1909, 76-2149
Residue removal
Water, 76-2122
Residues/food and feed
Vegetables, 76-1888
Residues/plants
Medicinals and condiments 76-1888
Toxicity/experimental animals
Crustacea, 76-2039
Insects, 76-2039
Treatment of poisoning
Atropine, 76-1931

Malathion derived compounds
Chromatography
Gas-liquid, 76-2090

Maleic anhydride
Immunology
In vitro, 76-2001

Maleic hydrazide
see also Herbicides
Chromosomes/genes
Plants, 76-1735
Mutagenesis/teratogenesis
Plants, 76-1735
Residue degradation
Soil, 76-2163
Residues/soil
Adsorption, 76-2163
Therapeutic use
Cancer, 76-2027

Maneb
see also Carbamates; Fungicides
Biotransformation
Plants, 76-2021
Residues/food and feed
Vegetables, 76-2190
Toxicity/humans
Occupational, 76-1704

MBT
see Methabenzthiazuron

MCPA
see also Herbicides
Factors influencing metabolism/ toxicity
Adaptation, 76-1990
Taxon, 76-1990
Residue degradation
Water, 76-2149
Toxicity/experimental animals
Microorganisms, 76-1990

Menazon
see also Organophosphates
Growth
Microorganisms, 76-1944
Photodecomposition, 76-1893

Mercurials
Distribution/storage
Pheasant, 76-1723
Environmental pollution, 76-1605 76-1606, 76-2105, 76-2106
Factors influencing metabolism/ toxicity
Structure/function, 76-2295
Morbidity and mortality statistics
Japan, 76-1680
Sweden, 76-1680
Residue degradation
General, 76-2127
Residues/air
Industrial, 76-2173
Residues/food and feed
General, 76-2181
Total diet, 76-1614
Fish, 76-2119
Residues/humans
Urine, 76-2173
Residues/non-target organisms
General, 76-2127
Birds, 76-1626, 76-2117
Duck, 76-1634
Fish, 76-1636, 76-2139
Seal, 76-2150
Residues/soil

Mercurials (cont'd)
General, 76-2181
Residues/water
Oceans/seas, 76-2139
Rivers/streams, 76-2139
Respiratory system
Human, 76-1699
Reviews
Monitoring and residues, 76-2110
Toxicology and pharmacology 76-2110
Toxicity/experimental animals
General, 76-2295
Toxicity/humans
Accidental, 76-1680
Occupational, 76-1699
Vision
Human, 76-2212

Metaldehyde
see also Molluscicide
Toxicity/non-target organisms
Dog, 76-1696

Metathion E-50
see Fenitrothion

Methabenzthiazuron
see also Herbicides
Biotransformation
Plants, 76-1814
In vitro, 76-1745
Distribution/storage
Plants, 76-1814
Factors influencing metabolism/ toxicity
Light/radiation, 76-1745

Methamidophos
Chromatography
Gas-liquid, 76-2097

Methoprene
see also Juvenile hormones and analogs
Biotransformation
Chicken, 76-2025
Chromatography
Gas-liquid, 76-2092
Distribution/storage
Chicken, 76-2024, 76-2025
Excretion
Chicken, 76-2024

Methyl benzimidazol-2-yl carbamate
see Carbendazim

Methyl bromide
see also Fumigants
Residues/soil
General, 76-1623

Methyl demeton
see also Organophosphates
Enzyme activity
Cholinesterase, 76-2208
Toxicity/humans
Intentional, 76-2208
Treatment of poisoning
Atropine, 76-2208

Methyl mercaptophos
see also Organophosphates
Prevention
Decontamination, 76-1937

Methyl parathion
see also Organophosphates
Analysis
Sample preparation, 76-2348
Biotransformation
In vitro, 76-2281
Chromatography
Thin-layer, 76-2074, 76-2348
Enzyme activity
Cholinesterase, 76-1940
Esterases, 76-1940
Residues/food and feed
Dairy products, 76-1663

Methylacetono-chlorophos-3
see also Organophosphates
Kidney
Animals/experimental, 76-1807
Liver
Animals/experimental, 76-1807
Nervous system
Animals/experimental, 76-1807

Methylmercury dicyandiamide
see also Fungicides
Biotransformation
Microorganisms, 76-1743
Factors influencing metabolism/toxicity
Adaptation, 76-1743

Metribuzin
see also Herbicides
Biotransformation
Plants, 76-1818, 76-1980
Residues/soil
Adsorption, 76-1667

Mevinphos
see also Organophosphates
Factors influencing metabolism/toxicity
Sex, 76-2318
Temperature, 76-2339
Toxicity/experimental animals
Gerbils, 76-2318

Meziranyl
see Aziprotryn

Mirex
see also Organochlorines
Biotransformation
In vitro, 76-1898
Monkey, 76-2243
Distribution/storage
Monkey, 76-2242, 76-2243
Enzyme activity
Mixed function oxidases, 76-2286
Excretion
Monkey, 76-2242
Factors influencing metabolism/

Mirex (cont'd)
toxicity
Sex, 76-2286
Residues/non-target organisms
General, 76-1630, 76-1632
Residues/soil
General, 76-1632

MO
see CNP

Molinate
see also Herbicides
Factors influencing metabolism/toxicity
Adaptation, 76-1990
Taxon, 76-1990
Toxicity/experimental animals
Microorganisms, 76-1990

Molluscicide
see Clonitralide; Fentin acetate; Metaldehyde

Monocrotophos
see also Organophosphates
Biotransformation
Insects, 76-2278, 76-2278
Enzyme activity
Cholinesterase, 76-1719
Factors influencing metabolism/toxicity
Humidity, 76-1719
Residues/food and feed
Fruits, 76-1892

Monox
see Fungicides

Monuron
see also Herbicides
Toxicity/experimental animals
Microorganisms, 76-2324

MSMA
see also Herbicides
Residue degradation
Soil, 76-2170

Nabam
see also Carbamates; Fungicides
Biochemical effects
General, 76-1947
Biotransformation
Plants, 76-2021
Liver
Animals/experimental, 76-1947

Naled
see also Organophosphates
Distribution/storage
Plankton/algae, 76-2236
Spectrometry
Atomic absorption/emission 76-1840
Toxicity/experimental animals
General, 76-1738
Birds, 76-1738
Fish, 76-1738
Insects, 76-1738
Reptiles, 76-1738

Naphthaleneacetic acid
see also Herbicides
Residue degradation
General, 76-2187

1,8-Naphthalic anhydride
see Protect

Neguvon
see Trichlorfon

Nematocides
see Dazomet; Telone

Nitralin
see also Herbicides
Electrometry
Polarography, 76-2064
Residue degradation
Soil, 76-2167

Nitrapyrin
see also Soil bactericide
Respiration, cellular
Microorganisms, 76-1742

Nitro compounds
Carcinogenesis
Human, 76-2219
Chromatography
Gas-liquid, 76-2085
Thin-layer, 76-1842
Laws and regulations
Germany (BRD), 76-2219
United Kingdom, 76-2219
USA, 76-2219

Nitrofen
see also Herbicides
Treatment of poisoning
General, 76-1600

Ordram
see Molinate

Organochlorines
see also Aldrin; BHC; Chlordane; Chlordecone; Chlordimeform; DDE; DDT; Dicofol; Dieldrin; Endosulfan; Endrin; Heptachlor; Lindane; Mirex; TDE; Toxaphene
Absorption
In vitro, 76-1981
Analysis
Sample preparation, 76-1846 76-2345
Biotransformation
Microorganisms, 76-2230
Plants, 76-2241
Carcinogenesis
Animals/experimental, 76-1761
Human, 76-1761, 76-1868
Chromatography
Column, 76-1839, 76-2073
Gas-liquid, 76-1844, 76-2345
Thin-layer, 76-1833, 76-2098
Economics, 76-1598
Environmental pollution, 76-1602 76-2102, 76-2108, 76-2109
Enzyme activity
General, 76-1808, 76-1808

Organochlorines (cont'd)
General, 76-1979, 76-1981
Mixed function oxidases, 76-2059
Excretion
Cow, 76-1766
Experimental design
Toxicology and pharmacology 76-1757, 76-1798
Factors influencing metabolism/toxicity
Interactions, 76-1766, 76-1975
Structure/function, 76-2321
Growth
Microorganisms, 76-1808
Laws and regulations
Japan, 76-1694
Lipids/steroids/sterols
Animals/experimental, 76-1987
Metabolism
Microorganisms, 76-1986
Morbidity and mortality statistics
Mexico, 76-1693
Mutagenesis/teratogenesis
Microorganisms, 76-1808, 76-2254
Prevention
Disposal, 76-1925
Residue degradation
Soil, 76-1644
Water, 76-2130
Residues/food and feed
General, 76-1625, 76-1670 76-1916, 76-2180, 76-2181 76-2194, 76-2195
Total diet, 76-2158
Animal feed, 76-1904
Dairy products, 76-1646, 76-1664 76-1669, 76-1869, 76-1886 76-1904
Fruits, 76-1864
Residues/humans
General, 76-1916
Adipose, 76-1611, 76-1868
Blood, 76-1868
Milk, 76-1615, 76-1886, 76-1904 76-2180, 76-2194, 76-2195
Residues/non-target organisms
Birds, 76-2117
Crustacea, 76-1631
Fish, 76-1631, 76-1872
Molluscs, 76-1631, 76-1872
Residues/plants
Medicinals and condiments 76-1863
Ornamentals, 76-1863
Residues/soil
General, 76-1644, 76-2181
Residues/water
General, 76-2130
Lakes/ponds, 76-2176
Respiration, cellular
In vitro, 76-1726
Microorganisms, 76-1808
Respiratory system
Human, 76-1699
Reviews
Monitoring and residues, 76-2107
Toxicology and pharmacology 76-1987
Safety standards
Acceptable daily intake, 76-1694
Spinal cord
In vitro, 76-1726
Toxicity/experimental animals
Birds, 76-1801
Rat, 76-2321
Toxicity/humans
General, 76-1936
Treatment of poisoning
General, 76-2059
Vision
Animals/experimental, 76-1979 76-1981

Organophosphates
see also Amine; Azinphosmethyl; BAY NTN 9306; Butonate; Chlorfenvinphos; Chlorpyrifos; Chlorpyrifos-methyl; Chlorthion; CKB 1028; Coumaphos; Cyanophos; Cystaphos; Demephion; DFP; Diazinon; Dichlorvos; Dimefox; Dimethoate Disulfoton; Ethephon; Ethion; Famphur; Fenitrothion; Fensulfothion; IDIMP; Inezin; Leptophos; Malathion; Menazon; Methyl demeton; Methyl mercaptophos; Methyl parathion; Methylacetonochlorophos-3; Mevinphos; Monocrotophos; Naled; Ovadophos; Paraoxon; Parathion; Phorate; Phosalone; Phosmet; Phosphamidon; Phospholan; Phoxim; Ronnel; Sarin; Soman; Temophos; Terbufos; Tetrachlorvinphos; Tri-*o*-cresyl phosphate; Trichlorfon; Trichlorometaphos-3; Trichlorophos; Triphenyl phosphate
Amino acids/peptides/proteins
Human, 76-1924
Analysis
General, 76-2067
Sample preparation, 76-1846
Biotransformation
Microorganisms, 76-2030
Plants, 76-2241
Chromatography
Gas-liquid, 76-1844, 76-2079 76-2085
Paper, 76-1827
Thin-layer, 76-1842, 76-1847 76-2082
Economics, 76-1598
EEG
Human, 76-1687, 76-2225 76-2227
Electrolytes
Human, 76-1682
Environmental pollution, 76-1602 76-2102, 76-2108
Enzyme activity
General, 76-1752, 76-1808
Cholinesterase, 76-1764, 76-1936 76-1941, 76-2206
Mixed function oxidases, 76-2059 76-2298
UDP-glucuronyl transferase 76-1961
Experimental design
Toxicology and pharmacology 76-1798
Factors influencing metabolism/toxicity
Structure/function, 76-1941
Growth
Microorganisms, 76-1808
Heart
Human, 76-1682
Kidney
Animals/experimental, 76-1939
Laws and regulations
Japan, 76-1694
Spain, 76-1918
Liver
Animals/experimental, 76-1939
Morbidity and mortality statistics
Hungary, 76-2205
Mexico, 76-1693
Mutagenesis/teratogenesis
Microorganisms, 76-1808
Nervous system
Human, 76-1924
Photodecomposition, 76-1612
Plasma/serum
Human, 76-1924
Prevention
Disposal, 76-1925
Residue dynamics, 76-1612
Residues/air
Industrial, 76-2115
Residues/food and feed
General, 76-1670, 76-2181
Meat, 76-1860
Vegetables, 76-1864
Residues/non-target organisms
Fish, 76-1657
Residues/plants
Medicinals and condiments 76-1863
Ornamentals, 76-1863
Residues/soil
General, 76-2181
Residues/water
Lakes/ponds, 76-1657
Respiration, cellular
In vitro, 76-1726
Microorganisms, 76-1808
Respiratory system
Human, 76-1699
Safety standards
Acceptable daily intake, 76-1694
Spectrometry
Colorimetry, 76-1849
Spinal cord
In vitro, 76-1726
Toxicity/experimental animals

Organophosphates (cont'd)
Birds, 76-1801
Toxicity/humans
General, 76-1936
Accidental, 76-1682, 76-2221
Occupational, 76-1687, 76-1699 76-1705, 76-1706, 76-1924 76-2199, 76-2206, 76-2221 76-2225
Toxicity/non-target organisms
General, 76-1918
Treatment of poisoning
General, 76-1600, 76-2059 76-2211
Atropine, 76-1928, 76-2199
Oximes, 76-1988
Pralidoxime, 76-2199
Vision
Animals/experimental, 76-2274

Oryzalin
see also Herbicides
Chromatography
Gas-liquid, 76-2089
Electrometry
Polarography, 76-2064

Osbac
see BPMC

Ovex
see also Organophosphates
Enzyme activity
Acid phosphatase, 76-1995
Factors influencing metabolism/ toxicity
Adaptation, 76-1995

Oxadiazon
see also Herbicides
Distribution/storage
Birds, 76-1714
Cow, 76-1714

Oxythioquinox
see also Fungicides
Residues/food and feed
Fruits, 76-2193

Paraoxon
see also Organophosphates
Enzyme activity
Cholinesterase, 76-1942
Lipids/steroids/sterols
Animals/experimental, 76-2046
Toxicity/humans
Intentional, 76-2201

Paraquat
see also Herbicides
Absorption
Rat, 76-1754
Chromatography
Thin-layer, 76-2083
Enzyme activity
General, 76-1712
Factors influencing metabolism/ toxicity
General, 76-1783

Paraquat (cont'd)
Interactions, 76-1712, 76-1767
Nutritional state, 76-1767
Lipids/steroids/sterols
Animals/experimental, 76-1767
Lung
Animals/experimental, 76-1713 76-1744, 76-1747, 76-1754 76-1783
Human, 76-1927, 76-1932 76-2197
Spectrometry
Colorimetry, 76-1841
Toxicity/experimental animals
Mouse, 76-1783
Plankton/algae, 76-1799
Rat, 76-1783
Toxicity/humans
Accidental, 76-1933, 76-2212
Intentional, 76-1922, 76-1927
Occupational, 76-1704
Treatment of poisoning
General, 76-1922
Bentonite, 76-1932
Fuller's earth, 76-1932
Hypoxia, 76-1982
Vision
Human, 76-1933, 76-2212

Parathion
see also Organophosphates
Absorption
In vitro, 76-1962
Analysis
Sample preparation, 76-2084 76-2348
Biotransformation
In vitro, 76-1728
Insects, 76-2278, 76-2278
Blood cells
Animals/experimental, 76-2302
Chromatography
Gas-liquid, 76-2084, 76-2097
Gel permeation, 76-1830
Thin-layer, 76-1836, 76-2061 76-2348
Electrolytes
Animals/experimental, 76-1711
Enzyme activity
Cholinesterase, 76-1929, 76-1931 76-2302
Mixed function oxidases, 76-1728 76-1945
Factors influencing metabolism/ toxicity
Interactions, 76-2053
Heart
Human, 76-2222, 76-2223
Hormones
Animals/experimental, 76-2053
Lipids/steroids/sterols
Animals/experimental, 76-2046
Human, 76-1929
Liver
Animals/experimental, 76-2053
Metabolism
Rat, 76-1722
Porphyrins

Parathion (cont'd)
In vitro, 76-1728
Residue degradation
Water, 76-1909, 76-2124, 76-2149
Residue dynamics, 76-2121
Residue removal
Plants, 76-1871
Residues/food and feed
Fruits, 76-2118
Residues/non-target organisms
Crustacea, 76-2121
Residues/soil
Volatilization, 76-2118, 76-2124
Respiration, cellular
In vitro, 76-1945
Respiratory system
Human, 76-2222
Spectrometry
Colorimetry, 76-1829
Thermoregulation
Animals/experimental, 76-1785 76-1785
Toxicity/humans
Accidental, 76-1929
Intentional, 76-2201, 76-2222 76-2223
Toxicity/non-target organisms
Dog, 76-1696
Treatment of poisoning
Atropine, 76-1929
Pralidoxime, 76-1929

Parathion derived compounds
Chromatography
Thin-layer, 76-2075
Liver
Animals/experimental, 76-1957

PCNB
see also Fungicides
Chromatography
Gas-liquid, 76-1838, 76-2071
Distribution/storage
Mouse, 76-1776
Rat, 76-1776
Electrometry
Polarography, 76-1838
Mutagenesis/teratogenesis
Animals/experimental, 76-1776
Residue degradation
Plants, 76-2185
Soil, 76-2184
Residues/food and feed
Vegetables, 76-2184
Spectrometry
Infrared, 76-1838
Toxicity/humans
Accidental, 76-2212
Vision
Human, 76-2212

PCP
see also Herbicides
Distribution/storage
Rat, 76-1966
Excretion
Rat, 76-1966

PCP (cont'd)
Residues/air
Industrial, 76-1633
Respiration, cellular
In vitro, 76-1954
Toxicity/experimental animals
Rat, 76-1965
Treatment of poisoning
General, 76-1600

Perfluidone
see also Herbicides
Residue degradation
Soil, 76-2171
Residues/soil
Movement, 76-2171

Petkolin
Enzyme activity
Acid phosphatase, 76-1995
Factors influencing metabolism/toxicity
Adaptation, 76-1995

Phamophos
see Famphur

Phenmedipham
see also Carbamates; Herbicides
Toxicity/experimental animals
Microorganisms, 76-2325

Phenylmercuric acetate
see PMA

Phenylmercury acetate
see PMA

Phorate
see also Organophosphates
Analysis
Sample preparation, 76-2062
Biotransformation
Microorganisms, 76-1984
Chromatography
Gas-liquid, 76-2062
Factors influencing metabolism/toxicity
Taxon, 76-2289
Metabolism
Helminths, 76-2289
Nitrogen fixation
Microorganisms, 76-2308
Residues/food and feed
Vegetables, 76-1896

Phosalone
see also Organophosphates
Enzyme activity
Acid phosphatase, 76-1995
Factors influencing metabolism/toxicity
Adaptation, 76-1995

Phosmet
see also Organophosphates
Absorption
Pig, 76-1812
Biotransformation
Pig, 76-1812
Rat, 76-2329
Blood/body fluids
Animals/experimental, 76-2327
Distribution/storage
Pig, 76-1812
Rat, 76-2329
Enzyme activity
Cholinesterase, 76-2327
Sympathetic nerves
Animals/experimental, 76-2327
Toxicity/experimental animals
Chicken, 76-2327
Fish, 76-1973
Rabbit, 76-2327

Phosphamide
see Dimethoate

Phosphamidon
see also Organophosphates
Biotransformation
In vitro, 76-2283, 76-2284
Enzyme activity
Esterases, 76-2283, 76-2284
Residues/plants
Forest, 76-1616
Respiration, cellular
In vitro, 76-2284

Phospholan
see also Organophosphates
Biotransformation
Insects, 76-2278
Excretion
Insects, 76-2278

Phospholine
see Organophosphates

Phosvel
see Leptophos

Phoxim
see also Organophosphates
Photodecomposition, 76-1859
Residues/plants
Medicinals and condiments 76-1859

Phthalophos
see Phosmet

Picloram
see also Herbicides
Factors influencing metabolism/toxicity
Humidity, 76-1907
Temperature, 76-1907
Residue degradation
Plants, 76-1880
Soil, 76-1880
Residues/plants
Forest, 76-2141
Residues/soil
Adsorption, 76-2156
Movement, 76-1624, 76-2141

Piperonyl butoxide
see also Synergists
Therapeutic use
Cancer, 76-2253

Piperonylate
see also Synergists
Biotransformation
Microorganisms, 76-2033

PMA
see also Fungicides; Herbicides
Amino acids/peptides/proteins
Animals/experimental, 76-2301
Distribution/storage
Rat, 76-2301
Enzyme activity
Mixed function oxidases, 76-2301
Growth
Microorganisms, 76-2238
Liver
Animals/experimental, 76-2301
Peripheral nerves
Animals/experimental, 76-2273
Respiration, cellular
Microorganisms, 76-2238
Toxicity/experimental animals
Microorganisms, 76-2238

Polychlorinated biphenyls
Biotransformation
Microorganisms, 76-1762
Chromatography
Column, 76-1839, 76-2073
Gas-liquid, 76-2100
Environmental pollution, 76-1601 76-2102
Enzyme activity
General, 76-1733
Factors influencing metabolism/toxicity
Interactions, 76-1733, 76-2316
Taxon, 76-2316
Growth
Microorganisms, 76-2299
Residue degradation
Water, 76-2159
Residues/food and feed
General, 76-1625
Total diet, 76-1614
Animal feed, 76-1876
Dairy products, 76-1876
Poultry, 76-1876
Residues/humans
Adipose, 76-1870
Milk, 76-1640, 76-1913
Residues/non-target organisms
Duck, 76-1634, 76-1637
Fish, 76-1636, 76-1912, 76-2139
Seal, 76-2150
Residues/water
Oceans/seas, 76-1877, 76-2139
Rivers/streams, 76-2139, 76-2140
Reviews
Monitoring and residues, 76-1857
Spectrometry
Colorimetry, 76-1825
UV, 76-1825

Potassium-2-formyl- 1,4-chlorophenoxyacetate
see also Herbicides
Factors influencing metabolism/toxicity

Potassium-2-formyl- 1,4-chlorophenoxyacetate (cont'd)
Interactions, 76-2316
Taxon, 76-2316

Pralidoxime
Plasma/serum
Human, 76-1958
Toxicity/humans
Experimental, 76-1958
Vision
Human, 76-1958

Prometryne
see also Herbicides
Mutagenesis/teratogenesis
Microorganisms, 76-2267
Residues/soil
Movement, 76-1666, 76-2169

Pronamide
see also Herbicides
Distribution/storage
Plants, 76-2332
Residues/soil
Adsorption, 76-2332

Propanil
see also Herbicides
Chromatography
Gas-liquid, 76-1848

Propham
see also Herbicides
Enzyme activity
Cholinesterase, 76-1772
GOT, 76-1772
GPT, 76-1772

Propoxur
see also Carbamates
Growth
Microorganisms, 76-2342

Propyzamide
Residue degradation
Soil, 76-1665, 76-2133

Protect
see also Herbicides
Analysis
Sample preparation, 76-2070
Chromatography
Gas-liquid, 76-2070

Pyramin
see Pyrazon

Pyrazon
see also Herbicides
Amino acids/peptides/proteins
Human, 76-1923
Enzyme activity
GPT, 76-1923
Lactic dehydrogenase, 76-1923
Liver
Human, 76-1923
Residues/soil
Adsorption, 76-2162
Skin
Human, 76-1923
Toxicity/experimental animals

Pyrazon (cont'd)
Microorganisms, 76-2325
Toxicity/humans
Occupational, 76-1923

Pyrethrin I
see also Botanicals; Pyrethrins
Chromatography
Gas-liquid, 76-2081

Pyrethrins
see also Allethrin; Pyrethrin I
Analysis
General, 76-2067
Environmental pollution, 76-1602
Residues/food and feed
General, 76-1661

Quinomethionate
see Oxythioquinox

Ro 10-3108
see also Juvenile hormones and analogs
Residue degradation
Water, 76-1900

Ro-Neet
see Cycloate

Rodenticides
see also Crimidine; Dicumarol; Strychnine; Thallium; Warfarin Zinc phosphide
Treatment of poisoning
General, 76-1679

Ronit
see Cycloate

Ronnel
see also Organophosphates
Absorption
Cow, 76-2319
Adrenal
Animals/experimental, 76-1951
Catecholamines
Animals/non-target, 76-1951
Digestive glands
Animals/experimental, 76-1805
Distribution/storage
Cow, 76-2319
Enzyme activity
Cholinesterase, 76-1951
Factors influencing metabolism/toxicity
Schedule of dosage, 76-2319
Growth
Animals/experimental, 76-1951
Liver
Animals/experimental, 76-1951
Vitamins/coenzymes
Animals/non-target, 76-1951

Rotenone
see also Botanicals
Enzyme activity
DNase, 76-2010
RNase, 76-2010
Growth
Animals/experimental, 76-2322
Microorganisms, 76-2322

Rotenone (cont'd)
Mitosis/meiosis
In vitro, 76-2042
Therapeutic use
Cancer, 76-2010
Toxicity/experimental animals
Plankton/algae, 76-2322

Sarin
see also Organophosphates
EEG
Animals/experimental, 76-1779
Enzyme activity
Cholinesterase, 76-1942

Sayphos
see Menazon

Schradan
see also Herbicides
Kidney
Animals/experimental, 76-1939
Liver
Animals/experimental, 76-1939

Sebumeton
see also Herbicides
Residues/soil
Adsorption, 76-2172

Sencor
see Metribuzin

Sevin
see Carbaryl

Silvex
see also Herbicides
Residues/food and feed
Fruits, 76-1873

Simazine
see also Herbicides
Bioassay, 76-1850
Factors influencing metabolism/toxicity
Schedule of dosage, 76-2040
Growth
Microorganisms, 76-2041
Nitrogen fixation
Microorganisms, 76-2294
Residue degradation
Soil, 76-1652, 76-2164
Residues/soil
Adsorption, 76-1652
Movement, 76-1652
Reviews
Monitoring and residues, 76-1910
Toxicity/experimental animals
Microorganisms, 76-2041, 76-2324

SMT
see Fenitrothion

Sodium trichloroacetate
see TCA

Soil bactericide
see Blasticidin S; Nitrapyrin

Soman
see also Organophosphates
Cytological effects

Soman (cont'd)
Animals/experimental, 76-1952
Enzyme activity
Cholinesterase, 76-1952
Kidney
Animals/experimental, 76-1952

Strychnine
see also Juvenile hormones and analogs; Rodenticides
Toxicity/non-target organisms
Dog, 76-1696

Succinic acid 2,2-dimethylhydrazide
see Daminozide

Sutan
see Butylate

Synergists
see also Piperonyl butoxide; Piperonylate
Environmental pollution, 76-1602
Enzyme activity
Mixed function oxidases, 76-2338
Estrogens
In vitro, 76-2338
Liver
In vitro, 76-2338

2,4,5-T
see also Dioxins; Herbicides
Kidney
Animals/experimental, 76-1778
Metabolism
Rat, 76-1778
Mutagenesis/teratogenesis
Animals/non-target, 76-1953
Residue degradation
Water, 76-2149
Residue dynamics, 76-1645
Residues/plants
Forest, 76-1645
Toxicity/non-target organisms
Cow, 76-1953
Plants, 76-1953

2,4,5 T derived compound
Chromatography
General, 76-1690
Skin
Human, 76-1690
Toxicity/humans
Occupational, 76-1690
Toxicity/non-target organisms
Chicken, 76-1690

TCA
see also Herbicides
Residues/food and feed
Cereals, 76-1618
Residues/water
Irrigation, 76-2166
Toxicity/experimental animals
Microorganisms, 76-2325

TCDD
see Dioxins

TDE
see also Organochlorines
Adrenal

TDE (cont'd)
Human, 76-1765
Chromosomes/genes
Animals/experimental, 76-2263
Mutagenesis/teratogenesis
Animals/experimental, 76-2263
Residues/food and feed
Animal feed, 76-2154
Dairy products, 76-1653
Residues/non-target organisms
Duck, 76-1637
Residues/water
Oceans/seas, 76-1877
Therapeutic use
Cushing's syndrome, 76-1759
76-1765

Tebuthiuron
see also Herbicides
Biotransformation
Dog, 76-2051
Duck, 76-2051
Fish, 76-2051
Rabbit, 76-2051
Excretion
Dog, 76-2051
Duck, 76-2051
Fish, 76-2051
Rabbit, 76-2051
Rat, 76-2051

Telone
see also Nematocides
Residues/soil
Adsorption, 76-1654
Movement, 76-1887

Temik
see Aldicarb

Temophos
see also Organophosphates
Enzyme activity
Cholinesterase, 76-1998
Growth
Microorganisms, 76-1993
Toxicity/experimental animals
Fish, 76-1991
Microorganisms, 76-2012
Plankton/algae, 76-1991, 76-2012

Tepa
see also Chemosterilants
Chromosomes/genes
Animals/experimental, 76-1770
Factors influencing metabolism/toxicity
Interactions, 76-1770
Mutagenesis/teratogenesis
Animals/experimental, 76-1770

Terbufos
see also Organophosphates
Residue degradation
Soil, 76-1883
Residues/food and feed
Vegetables, 76-1620

Terbutryne
see also Herbicides
Residues/soil
Adsorption, 76-2172

Tetrachloroisophthalonitrile
see Chlorothalonil

Tetrachlorvinphos
see also Organophosphates
Chromatography
Gas-liquid, 76-2346
Growth
Microorganisms, 76-2342

TFM
Toxicity/experimental animals
Crustacea, 76-2039
Insects, 76-2039

TH 6040
see Difluron

Thallium
see also Rodenticides
Cardiovascular system
Human, 76-2218
Kidney
Animals/experimental, 76-1939
Liver
Animals/experimental, 76-1939
Porphyrins
Human, 76-2210
Toxicity/humans
Accidental, 76-1691, 76-2218
Intentional, 76-1697, 76-2210
Toxicity/non-target organisms
Dog, 76-1696
Treatment of poisoning
General, 76-1683, 76-1691
Berlin blue, 76-1697
Mannitol, 76-1697
Propranolol, 76-2210

Thiabendazole
see also Fungicides
Analysis
Sample preparation, 76-1845
Photodecomposition, 76-1875
Spectrometry
UV, 76-1845, 76-2072

Thiophanate
see also Fungicides
Analysis
Sample preparation, 76-1845
Spectrometry
UV, 76-1845

Thiophanate-methyl
see also Fungicides
Residues/plants
Orchard, 76-2134
Skin
Human, 76-1707
Toxicity/humans
Occupational, 76-1707

Thiram
see also Fungicides
Amino acids/peptides/proteins
Animals/experimental, 76-2006

Thiram (cont'd)
Human, 76-2006
Bioassay, 76-2357
Biochemical effects
General, 76-1947
Blood/body fluids
Animals/experimental, 76-2005
Carcinogenesis
Microorganisms, 76-2256
Factors influencing metabolism/toxicity
Formulation, 76-2005
Fibroblasts
Animals/experimental, 76-2314
Human, 76-2314
Kidney
Animals/experimental, 76-2005
Liver
Animals/experimental, 76-1947 76-2005
Metabolism
Microorganisms, 76-2256
Mitosis/meiosis
In vitro, 76-2042
Residue degradation
Food and feed, 76-1911
Soil, 76-1878

Tillam
see Pebulate

Tin compounds
Respiratory system
Human, 76-1699
Spectrometry
Colorimetry, 76-2352
Toxicity/humans
Occupational, 76-1699

TMTD
see Thiram

Toxaphene
see also Organochlorines
Absorption
Fish, 76-2234

Tri-o-cresyl phosphate
see also Organophosphates
Amino acids/peptides/proteins
Animals/experimental, 76-2052
Immunology
Animals/experimental, 76-2052

Triarimol
see also Fungicides
Enzyme activity
Mixed function oxidases, 76-1780
Liver
Animals/experimental, 76-1780

Tributyl-3-chlorobenzylammonium bromide
see also Herbicides
Respiration, cellular
Microorganisms, 76-1742

Trichlorfon
see also Organophosphates
Absorption
Pig, 76-1812
Adrenal
Animals/experimental, 76-1792
Amino acids/peptides/proteins
Animals/experimental, 76-1789 76-1794
Behavior
Animals/experimental, 76-1793
Biochemical effects
General, 76-2323
Biotransformation
Pig, 76-1812
Blood cells
Animals/experimental, 76-1789 76-1803
Brain
Animals/experimental, 76-2269 76-2270, 76-2271
Carbohydrates
Animals/experimental, 76-1795 76-2270
Demyelination
Human, 76-1686
Digestive glands
Animals/experimental, 76-2315
Distribution/storage
Cow, 76-1795
Pig, 76-1812
Rabbit, 76-2323
Enzyme activity
General, 76-1773, 76-1795
Acid phosphatase, 76-2271 76-2315
ATPase, 76-2270
Cholinesterase, 76-1731, 76-1793 76-1803, 76-1940, 76-2271
Cytochrome oxidase, 76-2269 76-2272
Esterases, 76-1940
β-Glucuronidase, 76-2315
Succinic dehydrogenase, 76-2269 76-2272
Enzyme assay
Cholinesterase, 76-2354
Excretion
Sheep, 76-1811
Factors influencing metabolism/toxicity
Interactions, 76-1731
Fibroblasts
In vitro, 76-2057
Growth
Animals/experimental, 76-1793
Kidney
Animals/experimental, 76-1792
Liver
Animals/experimental, 76-1792
Metabolism
Cow, 76-1797
Musculoskeletal system
Animals/experimental, 76-1792
Nucleic acids
Animals/experimental, 76-2279
Pancreas (exocrine)

Trichlorfon (cont'd)
Animals/experimental, 76-2315
Plasma/serum
Animals/experimental, 76-1794 76-1803
Polyneuritis
Human, 76-1686
Respiration, cellular
Animals/experimental, 76-2270
Spectrometry
Colorimetry, 76-2354
Toxicity/experimental animals
Birds, 76-1813
Chicken, 76-1803
Toxicity/humans
Accidental, 76-1686, 76-1688
Treatment of poisoning
Atropine, 76-1810

Trichlorometaphos-3
see also Ronnel; Organophosphates
Digestive glands
Animals/experimental, 76-1805

Trichlorophos
see also Organophosphates
Behavior
Animals/experimental, 76-1804
Blood cells
Animals/experimental, 76-1803 76-1803
Enzyme activity
Cholinesterase, 76-1803
Skin
Animals/experimental, 76-1804
Toxicity/experimental animals
Chicken, 76-1803
Vision
Animals/experimental, 76-1804

Trifluralin
see also Herbicides
Electrometry
Polarography, 76-2064
Residue degradation
Soil, 76-2167

Triforine
see also Fungicides
Photodecomposition, 76-1639

Triphenyl phosphate
see also Organophosphates
Biotransformation
Insects, 76-1955
Enzyme activity
Esterases, 76-1955

Trolene
see Ronnel

Validamycin
see also Fungicides
Metabolism
Mouse, 76-2276
Toxicity/experimental animals
Rat, 76-2276

Vernam
see Vernolate

Warfarin
see also Rodenticides
Factors influencing metabolism/toxicity
Adaptation, 76-2011
Geographic location, 76-2011
Kidney
Animals/experimental, 76-1939
Liver
Animals/experimental, 76-1939
Spectrometry
UV, 76-2076

Zinc phosphide
see also Rodenticides
Toxicity/humans
Accidental, 76-1676, 76-1677

Zineb
see also Carbamates; Fungicides
Biochemical effects
General, 76-1947
Biotransformation
Plants, 76-2021
Fibroblasts
Animals/experimental, 76-2314
Human, 76-2314
Skin

Zineb (cont'd)
Human, 76-1930
Toxicity/humans
Occupational, 76-1930

Zolone
see Phosalone

Author Index

Ababei, L., 76-2331
Abe,E., 76-2225
Abe, J., 76-1706
Abou-Donia, M. B., 76-1777, 76-2000 76-2044
Abston, P. A., 76-2286
Achtzehn, M. K., 76-2175
Adamczewski, K. A., 76-1668
Adams, J. D., 76-2118
Adams, W. J., 76-1723
Addison, J. B., 76-1891
Aerts, F., 76-2042
Aharonson, N., 76-1892, 76-2134 76-2278
Ahdaya, S. M., 76-1785
Aherne, S. A., 76-1904
Aizicovici, H., 76-1651
Akhundov, K. F., 76-2012
Akhundov, V. Yu., 76-2012
Akin, F. J., 76-2027
Al-Doori, Z., 76-2260
Albonetti, G., 76-1736
Albright, L. J., 76-1631, 76-2140
Allen, S. H., 76-1802
Allen, T. J., 76-1851
Almeida, W. F., 76-1868, 76-1920 76-1953
Althaus, W. A., 76-1815, 76-2333
Alvarez, L. E., 76-1692
Ambe, Y., 76-2152
Ametov, A. S., 76-1759
Amir, D., 76-2047
Andersson, A., 76-1662
Andruszewska, A., 76-2341
Antze, H. H., 76-2147
Aoki, A., 76-1837
Appleby, A. P., 76-2172
Araki, Y., 76-1733
Araya, O., 76-1782
Archer, B. L., 76-1717
Archer, T. E., 76-1871
Arczynska, E., 76-1641
Arena, J. M., 76-1681
Arie, G., 76-1870
Arimatsu, N., 76-2275
Arimatsu, T., 76-1707
Arjmand, M., 76-2088
Armas, M. R., 76-2210
Artemenko, E. M., 76-1943
Asaf Ali, K., 76-1914
Asanuma, N., 76-1650
Asanuma, S., 76-1648
Ascher, K. R. S., 76-2278
Astolfi, E., 76-1611
Atkinson, B., 76-2199
Atkinson, G., 76-2265
Audley, B. G., 76-1717
Aulicino, F., 76-2257
Aust, S. D., 76-1767
Averitt, W. K., 76-1906
Avery, M. J., 76-1629
Ayrumyan, V. A., 76-1793, 76-1794 76-1795
Baba, H., 76-1837
Babina, E. Ya., 76-1795
Bachmann, F., 76-2076
Bahga, H. S., 76-2007
Bajgar, J., 76-1972
Bak, W., 76-1709
Bakke, J. E., 76-2034
Baldwin, F. L., 76-1666, 76-2169
Ballog, O., 76-1924
Banasiak, L., 76-1820
Banerjee, A. K., 76-2009
Bankowska, J., 76-1763
Banks, W. A., 76-1630
Barannyk, V. P., 76-2112
Barbuta, R., 76-1651
Barna, J., 76-2248
Barraquero, M. J. C., 76-1656
Bartell, C. K., 76-2115
Barton, S. J., 76-2089
Baskett, T. S., 76-1802
Bauza, C. A., 76-1615
Baxter, R. A., 76-1762
Beard, W. E., 76-1907
Becak, W., 76-2264
Belcher, D. H., 76-1728
Bellin, J., 76-2196
Bellward, G. D., 76-1997
Ben-Aziz, A., 76-2134, 76-2278
Bencivenga, B., 76-1646
Benedek, P., 76-2339
Benijts-Claus, C., 76-1799
Bennett, G. W., 76-2131
Benson, W. W., 76-1633, 76-2117
Berezovskiy, M. Ya., 76-1880
Berg, J. W., 76-1761
Berge, H., 76-2074, 76-2075
Bermejillo, M., 76-1918
Berndt, W. O., 76-1778
Bernhard, N. R., 76-1780
Beroza, M., 76-2066
Bertling, D., 76-1885
Besson, J., 76-1600
Betts, P. M., 76-1895
Bevenue, A., 76-1632
Bhatia, S. C., 76-2048
Bhattacharrya, P., 76-2010
Bhattacharya, S., 76-2008
Bicz, W., 76-2268, 76-2270
Biczowa, B., 76-2268, 76-2270
Bidleman, T. F., 76-1828
Bignami, M., 76-2257, 76-2258
Bimber, D. L., 76-2277
Birk, Y., 76-1897
Blair, A., 76-2063
Blair, D., 76-2261, 76-2261
Bluethgen, A., 76-1886
Boening, R. W., 76-2277
Boghianu, L., 76-1773
Bolt, H. M., 76-2338
Bontoyan, W. R., 76-2095
Borkowska, G., 76-1709
Botre, C., 76-1646
Boucard, M., 76-2321
Bourke,J. B., 76-2128
Bourne, S., 76-2096
Bradway, D. E., 76-1719
Brady, H. A., 76-2141
Bratanov, V., 76-1811
Breccia, A., 76-1736
Bressolle, F., 76-1862
Brewerton, H. V., 76-2107
Bridges, B. A., 76-2249
Brighenti, F., 76-2059
Brock, D. W., 76-2117
Brodie, J., 76-2011
Brooker, M. P., 76-2220
Brooks, G. T., 76-2282
Brown, A. W. A., 76-1821
Brown, J. R., 76-1963
Brown, M. J., 76-1896
Brownrigg, J. T., 76-1825
Bruk, A. A., 76-2351
Bruzzone, M. L., 76-2254
Buchanan, G. A., 76-1624
Buchenauer, H., 76-1639
Budimir, M., 76-1716
Budoi, G., 76-1644
Budrin, Yu. P., 76-2218
Budy, A. M., 76-1967
Bulkley, R. V., 76-1638
Bull, D. L., 76-2022
Burchfiel, J. L., 76-1779
Burkatskaya, E. N., 76-1700
Burns, K. A., 76-1985
Burrage, R. H., 76-2037
Bus, J. S., 76-1767, 76-1783
Buselmaier, W., 76-2262
Buys, M., 76-1714
Cagen, S. Z., 76-1783
Cambier, J., 76-2203
Campbell, C. J., 76-1896
Campo, M., 76-1845, 76-1863, 76-2072
Cardamone, G., 76-2254
Carere, A., 76-2254, 76-2257
Carlson, W. C., 76-2332
Carpenter, S., 76-1739
Carrington, C. B., 76-2197
Carruthers, I. B., 76-1717
Carter, C. H., 76-2167
Casalis, J., 76-2071
Casarett, L. J., 76-1965, 76-1966
Cash, G. A., 76-2154
Cegla, U. H., 76-1747
Chandra, P., 76-2233
Chapman, P. J., 76-2230
Chasson, A. L., 76-1719
Chen-kai, L., 76-2179
Cheng-jui, Q., 76-2179
Chervyakov, D. K., 76-1805
Ching, S. A., 76-1965, 76-1966
Chiu, M., 76-1712
Chkanikov, D. I., 76-1943
Chlebowski, J. F., 76-2030

Choi, K. L., 76-2125
Chow, I., 76-2196
Chow, L. Y., 76-1963
Chow, P. N. P., 76-1618
Clapp, D. W., 76-1908
Clark, A. G., 76-2281
Clayton, R., 76-2209
Cochrane, W. P., 76-1873
Cocisiu, M., 76-1651
Cockerill, A. F., 76-2089
Cockson, A., 76-2120
Colbert, F. O., 76-2172
Cole, E. R., 76-2076
Coleman, J. E., 76-2030
Colizzi, A., 76-2251
Collet, G. F., 76-1745, 76-1814
Comes, R. D., 76-2166
Conti, G., 76-2257
Conti, L., 76-2258
Cook, G. H., 76-2090
Cooper, J. E., 76-1813
Cooper, P., 76-1928
Copeland, M. F., 76-1776
Corley, C., 76-2028
Cortina de Luna, J. L., 76-1765
Cottrell, A., 76-2127
Coulston,F., 76-1894
Courtney, K. D., 76-1776
Cox, D. C., 76-1905
Cox, J. H., 76-1721
Craven, A. C. C., 76-2282
Crawford, M. J., 76-1727
Cremer, H. D., 76-2111
Cristea, E., 76-1773
Cropp, P. L., 76-2281
Cross,C., 76-1712
Crowder, L. A., 76-2234
Cucos, S., 76-1870
Cugurra, F., 76-2119
Cunat, P., 76-2143
Cunningham, W. L. Jr., 76-2050
Curl, E. A., 76-2029
Cutler, J. R., 76-1598
D'Arca, S. U., 76-2151
Dahlgren, S. E., 76-1889
Dahm, P. A., 76-1620, 76-1883
Dally, S., 76-2203
Daneshvar, H., 76-1992
Danon, J. M., 76-1739
Dasgupta, F. K., 76-2064
Data, O., 76-1674
Datta, K. K., 76-1996, 76-2233
Datta, N. P., 76-1910
David, D., 76-1969
Davidson, J. M., 76-1666, 76-2169
Davis, M., 76-1753
Davis, P. J., 76-1929
Davison, K. L., 76-1721, 76-2024
De Almeida, P. R., 76-1867
De Brabander, M., 76-2042
de Jong, L. P. A., 76-1988
De Moor, H., 76-1766
De Vargas, G. Z., 76-2158
Deagen, J., 76-2285
Dean, B. J., 76-2261
Dedek, W., 76-2086, 76-2279
Degraeve, N., 76-2251
Delak, M., 76-1810
Delatour, P., 76-1691
DeMatteis, F., 76-1957
Demint, R. J., 76-2166
Deng, C. B., 76-1963
Denys, A., 76-1774
Derache, R., 76-1864
Deshmukh, V. A., 76-2040, 76-2041
Desi, I., 76-2244
Deubert, K. H., 76-2121
Di Giuseppe,G., 76-2254
Di Pietro, A. M., 76-1736
Dikshith, T. S. S., 76-1996, 76-2233
Dinu, I., 76-1773
Dodd, J. D., 76-1851
Doguchi, M., 76-1614
Dollar, A. M., 76-1905
Domanski, J. J., 76-1719
Donchev, D., 76-1952
Dorn, S., 76-1900
Dorough, H. W., 76-1722, 76-2016
Douze, J. M. C., 76-2202
Downey, W. K., 76-1904
Dragomirescu, A., 76-2331
Dube, D. K., 76-2010
Dubovoy, V. P., 76-1943
Duffy, F. H., 76-1779
Dyck, P. J., 76-2045
Eberhardt, J., 76-1735
Ecke, G. G., 76-2020
Ecobichon, D. J., 76-1971
Eder, G., 76-1877
Edwards, R. W., 76-2220
Edwards,W. M., 76-1899
Efremov, M. I., 76-1806
Egert, G., 76-2256, 76-2267
Eisenbrand, G., 76-2240
El Zorgani, G. A., 76-1617
Eldredge, J. D., 76-2356
Eljasz, L., 76-1688
Elliston, J. O., 76-1802
Elzeftawy, A., 76-1655
Emmerson, J. L., 76-1780
Endecott, B. R., 76-2046
Endre, C., 76-2245
Engebretson, K. A., 76-1721
Engelhardt, G., 76-1977
Engst, R., 76-2174, 76-2247
Ermolenkov, V. V., 76-1658
Ernst, W., 76-2145
Esaki, H., 76-1706
Eto, M., 76-1955
Evans, E. S., Jr., 76-2237
Evans, J., 76-1782
Evdokimov, E. S., 76-2013
Faff, J., 76-1709
Farah, M., 76-1830, 76-1836
Farkas, I., 76-2244
Fassbender, H. W., 76-1660
Faudemay, F., 76-1947
Fedorov, V. V., 76-2002
Fennis, J. F. M., 76-1683
Ferguson, B. B., 76-1861
Fernandez, N. R., 76-1711
Ferrando, R., 76-1768, 76-1983
Filip, Z., 76-1808
Filippone, E. J., 76-1751
Fincher, R. C., 76-1725
Finlayson, D. G., 76-1896
Fischer, G. W., 76-2279
Fishbein, L., 76-2049
Fiveland, T. J., 76-2004
Fleeker, J. R., 76-1895
Floyd, G. L., 76-1993
Flynn, M. P., 76-1904
Fogg, G. E., 76-1956
Folk, E. D., 76-2318
Folmar, L. C., 76-2232
Fonnum, F., 76-1942
Ford, E. J. H., 76-1782
Forgash, A. J., 76-1998
Foster, T. S., 76-2017
Fournier, E., 76-2203
Franco, R., 76-1751
Frank, P. A., 76-2166
Frank, R., 76-2150
Freeman, H. P., 76-1899
Freidenberg, L., 76-1673
Fritz, J. S., 76-1629, 76-2094
Frolov, B. A., 76-2320
Fryer, G., 76-2200
Fuermaier, B., 76-2214
Fujimoto, Y., 76-2192
Fujita, K., 76-2212
Fukami, J. I., 76-1962
Fukano, S., 76-1614
Furie, B., 76-1935
Furman, D., 76-1656
Furuhashi, A., 76-2259
Furutani, F., 76-2182
Gabica, J., 76-1633
Gaeta, R., 76-1921
Gak, J. C., 76-1983
Gallo, M. A., 76-1714
Galobardes, J., 76-2097
Gangstad, E. O., 76-1906
Ganguli, S., 76-2009
Garcia Fernandez, J. C., 76-1611
Gardiner, T. H., 76-1754
Gatiatullin, N. N., 76-1807
Gattavecchia, E., 76-1736
Gautier, F., 76-1768
Gavica, J., 76-2117
Gazaryan, V. S., 76-2353
Gehring, P. J., 76-1784
Gencik, A., 76-1974
George, D. A., 76-1882
Geuens, S., 76-2042
Gevorkyan, S. G., 76-2343
Ghiasuddin, S. M., 76-1724
Ghilino, S., 76-1710
Gibson, J. E., 76-1767, 76-1783
Giddings, C. W., 76-1895
Gilbert, B. N., 76-2026, 76-2092
Gilbert, P. E., 76-1762
Gillespie, D. M., 76-2356
Gillott, M. A., 76-1993
Gimenez, E. R., 76-1711
Gingell, R., 76-2337
Giurgea, R., 76-1951
Glass, B. L., 76-1899

Glogowski, K., 76-1642
Glotfelty, D. E., 76-1899
Gmaz-Nikulin, E., 76-2003
Goforth, W. R., 76-1802
Golab, T., 76-1815, 76-2333
Golberg, L., 76-1894
Goldberg, M. C., 76-2153
Goldwater, L. J., 76-2105
Golovleva, L. A., 76-1990
Gomez Artero, J., 76-1834
Goodwin, J. T., 76-1738
Gorbacheva, N. A., 76-2350
Gordon, J. A., 76-1725
Gosteva, N. A., 76-2351
Goswami, B. B., 76-2010
Gotelli, C. A., 76-1833
Goto, T., 76-1706
Gottschewski, G. H. M., 76-1758
Goursaud, J., 76-2071
Goyena, R., 76-2122
Grace, H. K., 76-1894
Grafius, M. A., 76-1960
Grahneis, H., 76-2175
Graillot, C., 76-1983
Granovskaya, A. M., 76-1759
Grant, B. F., 76-2231
Grant, D. L., 76-1999
Grasblum, M., 76-2108
Grassi, N., 76-1866
Graul, E. H., 76-2219
Gray, R. S., 76-2121
Grechka, G. A., 76-1789, 76-1790 76-1791
Green, M. H. L., 76-2249
Green, V. A., 76-1936
Greenhalgh, R., 76-1873, 76-1970
Greim, H., 76-2256, 76-2267
Greve, P.A., 76-2144
Griffaton, G., 76-1947
Gripenberg, U., 76-2252
Groch, L., 76-1973
Gromisz, Z., 76-1755
Guardigli, A., 76-1714
Guenzi, W. D., 76-1907
Gugles, E., 76-1651
Guilbert-Ber, E., 76-1774
Guilhon, J., 76-1959
Gumennyy, V. S., 76-1698
Gunther, F. A., 76-2118
Gupta, P. K., 76-1994
Gupta, S. K., 76-2206
Gupta, S. P., 76-1734
Gutensohn, G., 76-2208
Guthrie, F. E., 76-1719, 76-1785
Haase, H. H., 76-2175
Hagiwara, A., 76-1648
Hajek, B. F., 76-1624
Halangk, W., 76-1938
Hallett, D. J., 76-1970
Hamelle, G., 76-1862
Hamm, K., 76-1964
Hammouda, M. H. M., 76-2325 76-2326
Hanneman, G. D., 76-2318
Hansen, C. R., Jr., 76-2039
Hanya, T., 76-2152
Haque, R., 76-2285
Hara, K., 76-1706
Harbison, J. W., 76-1684
Hargrave, B. T., 76-2137
Hasegawa, G., 76-1712
Hashimoto, Y., 76-1955
Hayashi, M., 76-1701
Hayashi, S., 76-1647, 76-1701
Hayes, J. D., 76-1737
Hearn, C. E. D., 76-2198
Hearnsberger, A. P., 76-1901
Heath, R. G., 76-1637, 76-1801
Heenchen, W., 76-1886, 76-2102
Heijbroek, W., 76-1594
Helrich, K., 76-2128
Helweg, A., 76-2163
Henneberg, M., 76-1946
Henriet, J., 76-1838
Henry, N., 76-1768
Herbold, B., 76-2262
Hermann, R. S., 76-1823
Hertwig, K., 76-1866
Herzel, F., 76-2146
Heuer, B., 76-1897
Heurteaux, P., 76-1657
Heyndrickx, A. M., 76-2091 76-2101
Hiebsch, C., 76-1938
Hietanen, E., 76-1718
Highland, H. A., 76-1661
Hikichi, M., 76-2023
Hill, E. F., 76-1801
Hiltbold, A. E., 76-1624
Hinderer, R. K., 76-2283 76-2284
Hiramatsu, R., 76-2182
Hirayama, H., 76-1706
Hitaka, K., 76-1948
Hoben, H. J., 76-1965, 76-1966
Hockwin, O., 76-1979, 76-1981
Hoebeke,J., 76-2042
Hoffman, D. G., 76-1780, 76-2051
Hoffman, R. D., 76-1626
Holdrinet, M., 76-2150
Holland, P., 76-1958
Holmes, D. S., 76-1727
Holmstead, R. L., 76-1898
Holusa, R., 76-1744
Hom, R., 76-2103
Hopen, H. J., 76-2332
Horiguchi, Y., 76-1702, 76-2225
Hornig, A. W., 76-1825
Houkam, E. G., 76-1695
Houkin, F. C. G., 76-2043
Houbrechts, N., 76-2251
Houff, S. A., 76-1684, 76-2045
Hrdlicka, J., 76-1957
Hristodorova, V., 76-1847
Hsu, K., 76-1954
Huang, J. C., 76-2138
Humuler, H., 76-2250
Hunt, L. M., 76-2026, 76-2092
Hussey, H. H., 76-1677
Huste, R. L., 76-1595
Hutson, D. H., 76-1727
Hutton, C. J., 76-1929
Hutzinger, O., 76-1853, 76-2018
Huyghebaert, A., 76-1766
Hylin, J. W., 76-1632
Ichim, A., 76-1773
Ide, H., 76-1648, 76-1702, 76-2225
Igarashi, H., 76-2185
Igarashi, M., 76-2188, 76-2189, 76-2192
Igata, A., 76-2213
Illes, S., 76-1863, 76-1863, 76-2072
Illyes, S., 76-1951
Inoue, T., 76-1852
Inoue, Y., 76-1706
Inscoe, M. N., 76-2066
Iranyi, J., 76-1686
Irvine, J., 76-1889
Isaacs, E., 76-2045
Ishige, T., 76-1704
Ishizuka, K., 76-1715
Ismail, M. A., 76-1890
Ismail, R., 76-1979, 76-1981
Ito, S., 76-1701
Ito, T., 76-1772
Ivey, M. C., 76-1621, 76-2031
Ivie, G. W., 76-2022
Iwata, Y., 76-2118
Izumiyama, T., 76-1703
Izurieta, E. M., 76-1711
Ja, B. Y., 76-1826
Jablonski, L., 76-2057
Jackson, R. S., 76-1684
Jacob, J. C., 76-1929
Jakubowska, B., 76-2342
Jamet, P., 76-2162
Jan, J., 76-1915
Jancarik, V., 76-1858
Jancso, H. E., 76-1860
Jansen, A. P., 76-1683
Jarczyk, H. J., 76-1745
Jeng, S. S., 76-1872
Joglekar, V. D., 76-2082
Johnson, A. W., 76-1602
Johnson, J. H., 76-2096
Johnson, J. M., 76-1905
Johnson, R. D., 76-1635
Johnson, W. F., 76-2089
Jolivet, G., 76-1959
Joly, E., 76-1600
Jonczyk, H., 76-1641
Jones, D., 76-2018, 76-2150
Joshi, O. P., 76-1910
Jouvenaz, D. P., 76-1630
Jovanovic, D., 76-1849
Jozsef, L., 76-2245
Juarez Fernandez, C., 76-1765
Juarez, M. B., 76-1611
Jung, H. D., 76-1930
Jung, K. D., 76-1798
Junk, G. A., 76-1629, 76-2094
Juszkiewicz, T., 76-1640
Kaars Sijpesteijn, A., 76-2036
Kaburaki, Y., 76-2345
Kacmar, P., 76-2224
Kada, T., 76-2259
Kadnikov, V. V., 76-2099
Kadoum, A. M., 76-2032
Kaiser, T. E., 76-1634

Kalashnykova, Yu. S., 76-2112
Kalinin, V. A., 76-1893
Kalra, R. L., 76-2038
Kamata, K., 76-1703
Kamataki, T., 76-1728
Kanamaru, N., 76-1627, 76-1628
Kani, T., 76-1614
Kapoor, I. P., 76-2130
Kapoor, S. K., 76-2038
Kapoor, V., 76-1994
Kappas, A., 76-2255
Karas, G.G., 76-2050
Karasek, F. W., 76-1853
Karel, A. K., 76-2235, 76-2280
Karpati, G., 76-1739
Kasai, A., 76-1648
Kashiwa, T., 76-2187, 76-2346, 76-2347
Kashyap, S. K., 76-2206
Kassel, H., 76-2338
Kato,H., 76-1705
Kato, K., 76-2259
Kato, T., 76-1705
Katz, M., 76-2084
Kawahara, T., 76-2188, 76-2189
76-2193
Kawale, G. B., 76-2082
Kawatski, J. A., 76-2039
Kazano, H., 76-2187
Ke-ming, C., 76-2179
Kearney, P. C., 76-2132
Keeler, P. A., 76-1784
Keeley, P. E., 76-2167
Kellogg, R. L., 76-1638
Kempny, R. S. G., 76-1610
Kentikyan, M. L., 76-1792, 76-1793
Keogh, R. G., 76-2266
Kern, A. D., 76-2336
Kesten, E. M., 76-1610
Keszthelyi, C.P., 76-2064
Ketchersid, M. L., 76-2171
Khan, M. A., 76-1857
Khan, S. U., 76-2017, 76-2093
Khaylovich, Yu. A., 76-1848
Khologov, I. Ya., 76-2320
Khomenko, L. I., 76-1937
Khurie, A. Zh., 76-1893
Kiely, M., 76-1844
Kilgore, L. T., 76-1901
Kimmerle, G., 76-1950
Kimura, M., 76-1701
Kirchner, K., 76-2074, 76-2075
Klawe, Z., 76-1641
Klein, W., 76-1979, 76-1980, 76-1981
76-2035, 76-2109
Klemmer, H. W., 76-1967
Kljajic, R., 76-1716
Klochkova, G. M., 76-2013
Klose, R., 76-2208
Klosterkoetter, S., 76-1608
Knyazev, A. F., 76-1937
Kobayashi, A., 76-2186
Kobayashi, S., 76-1703
Kochkurova, V. N., 76-1806
Kociba, R. J., 76-1784
Koehler, C. S., 76-2104
Kohli, J., 76-2035
Kohli, K. K., 76-2048
Kokordak, J., 76-2224
Kolesar, D., 76-1923, 76-1924
Kolov, A. Q., 76-1805
Komar, M., 76-1915
Komiyama, S., 76-1934
Kontek, B., 76-1642
Kontek, M., 76-1687, 76-2207
Kontson, A., 76-2132
Konyukhov, A. F., 76-2329
Kool, H. J., 76-1800
Koransky, W., 76-1823
Korte, F., 76-1876, 76-1980, 76-1981
76-2035, 76-2109
Korte, I., 76-1979
Koschier, F. J., 76-1778
Kosuge, T., 76-2019
Kothari, R. M., 76-1960
Kozler, M., 76-1689
Kozlov, Yu. S., 76-1937
Kozyntseva, P. V., 76-2001
Krampl, V., 76-1975
Krause, W., 76-1964
Krausz, T., 76-2244
Kreitzer, J. F., 76-1801
Kreuzer, W., 76-1669
Krishnamoorthy, K.K., 76-1879
76-1914
Kroidl, R. F., 76-1747
Krolikowska-Prasal, I., 76-1732
Kronberger, H., 76-1747
Krumm, M., 76-2173
Krumzdorov, A. M., 76-2014
Krylova, A. N., 76-2352
Krzyszton-Przekop, T., 76-1688
Kucsera, K., 76-2244
Kuhr, R., 76-2241
Kujawa, M., 76-2174, 76-2247
Kumar, D., 76-1654
Kumar, N. V. N., 76-2061
Kumarasamy, R., 76-1878
Kunz, S. E., 76-2031
Kupfer, D., 76-1987
Kurokawa, S., 76-1701
Kurosawa, K., 76-1648, 76-1650
76-2225
Kurotani, S., 76-1703
Kuthan, A., 76-1865
Kutsenko, S. A., 76-1731
Kutty, K. M., 76-1929
Kuul', A. A., 76-2329
Kuul', A. K., 76-2327
Kuwayama, T., 76-1837
LaFleur, K. S., 76-2155
Lahmann, E., 76-2146
LaHue, D. W., 76-2032
Laithwaite, J. A., 76-1922
Lakshminarayana, V., 76-2098, 76-2348
Lang, F., 76-1991
Lang, K., 76-1670
Lantseva, G.V., 76-1789
Laseter, J. L., 76-2115
Laska, A. L., 76-2115
Laurent, M., 76-1714
Laveglia, J., 76-1883
Lavigne, D. M., 76-2150
Lavon, U., 76-2047
Le Patourel, G. N. J., 76-1986
Leberl, C., 76-1823
Lee, I. N., 76-1715
Lee, K. S., 76-2093
Lee Lin, M. C. M., 76-1728
Lefar, M. S., 76-1714
Leiderman, L., 76-1866
Leistra, M., 76-1665
Lemberg, A., 76-1711
Leoni, V., 76-2151
Lerer, M. A. L., 76-1595
Lessa, J. M. M., 76-2264
Levy, J., 76-1774
Lewerenz, H. J., 76-2247
Lewis, G. C., 76-1908
Lidgett, R. A., 76-1762
Lignowski, E. M., 76-2332
Lijinsky, W., 76-2239
Linscott, D. L., 76-2128
Lipparini, L., 76-1604
Lishenko, I. D., 76-1787
Lishenko, N. P., 76-1787
Liska, D., 76-1924
Litov, R., 76-1712
Livingston, J. M., 76-1738
Lobato dos Santos, C. A., 76-1866
Lofgren, C. S., 76-1630
Lohs, K., 76-2279
Loomis, M., 76-2117
Looney, N. E., 76-1873
Lopez Capont, F., 76-1605
Lorgue, G., 76-1691
Lotti, M., 76-2059
Lovejoy, Jr., F. H., 76-1931
Lowy, R., 76-1947
Lozhkin, E. F., 76-2160
Lu, P. Y., 76-2130
Lubkowitz, J. A., 76-2097
Luk'yanchuk, V. D., 76-2006
Lund-Karlsen, R., 76-1942
Luquet, F., 76-2071
Lutz-Ostertag, Y., 76-1949
Luzhnikov, E. A., 76-1682
Lyr, H., 76-1820
MacGregor, A., 76-2106
Machimura, N., 76-1888
Macholz, R. M., 76-2174, 76-2247
MacNeil, J. D., 76-2023
Madaric, A., 76-2135, 76-2142, 76-2355
Mahoney, M. D., 76-1817, 76-2334
76-2335
Mahr, U., 76-2263
Maier-Bode, H., 76-2148
Maier, D., 76-2176
Mainprize, J. H., 76-1762
Maitlen, J. C., 76-1882
Majumdar, J. C., 76-1599
Majumdar, S. K., 76-1751
Majumder, S. K., 76-2061
Makarova, L.F., 76-2246
Makeev, A. M., 76-1943
Maki, A. W., 76-2236
Maksimova, E. P., 76-1788
Maksimovic, M., 76-1941
Malecki, R. A., 76-1802

Malepszy, S., 76-1735
Malik, J. K., 76-2007
Mallen, D. N. B., 76-2089
Maluszynski, M., 76-1735
Mamedova, A. P., 76-2012
Mancini, G., 76-1646
Manciulea, S., 76-1951
Mann, H. D., 76-2031
Mansell, R. S., 76-1655
Manske, D. D., 76-1635
Manyai, S., 76-1945
Marchand, A., 76-1959
Marcinkowska, B., 76-1687, 76-2207
Mariscal Mejias, M., 76-1765
Marriage, P. B., 76-2164
Marsh, J. R., 76-2136
Marshall, T. C., 76-2016
Martens, F. K., 76-2101
Martens, M. A., 76-2101
Martens, R., 76-2055
Martin, N. A., 76-2129
Martinez, A. J., 76-1684, 76-2045
Marvel, J. T., 76-2080
Maslennikova, V. I., 76-1796
Masud, S. Z., 76-2062
Mathew, C., 76-2260
Matsumoto, M., 76-1912, 76-1948
76-2073
Matsunaga, A. K., 76-1826, 76-2078
Matsushima, F., 76-1702
Matsushita, T., 76-1707
76-2275
Mattras, H., 76-1862
Mattsson, S., 76-1662
Matvienko, I. N., 76-2330
Matvienko, L. N., 76-1788
Maura, G., 76-2119
McClure, V. E., 76-2100
McConnell, J. K., 76-2046
McCormick, A., 76-2158
McDonough, L. M., 76-1882
McKenry, M. V., 76-1887
McKetta, John J., 76-1606
McLeod, J. E., 76-1818
Mecs, I., 76-1939
Meggitt, W. F., 76-1816, 76-2168
76-2336
Meguro, Y., 76-1647
Meisch, M. V., 76-2237
Meisner, J., 76-2278
Mel'nikov, N. N., 76-2054
Mendoza, C., 76-1944
Mendoza, C. E., 76-1775, 76-1781
76-1999
Menon, P. K., 76-2098
Menzel, D. B., 76-2000
Menzer, R. E., 76-1724, 76-2128
76-2283, 76-2284
Merenyuk, G. V., 76-1822
Merkle, M. G., 76-2171
Mesaki, T., 76-2346
Meshcherskaya, K. A., 76-2218
Mestres, R., 76-1657, 76-1845, 76-1863
76-2072
Metcalf, R. L., 76-2130
Metelitsa, V. K., 76-1797
Mi, M. P., 76-1967
Midio, A. F., 76-1842
Miettinen, J. K., 76-2110
Mihailescu, L., 76-1773
Mikhaylenko, V. K., 76-1789
Mikuni, I., 76-1933
Millar, D. B., 76-1960
Miller, J. H., 76-2167
Miller, R. W., 76-2028
Milohnoja, M., 76-1915
Miltenburger, H. G., 76-2263
Minagawa, N., 76-1647
Minear, R. A., 76-2136
Mirra, A. P., 76-1870
Miyake, Y., 76-1772
Miyamoto, H., 76-1955
Mizutani, T., 76-1948
Mollenhauer, H. P., 76-1917
Mollet, M. F., 76-1869
Monroe, R. J., 76-1719
Montgomery, R. R., 76-2204
Moore, B. J., 76-1743
Moore, J. C., 76-2090
Morelli, M. A., 76-2287
Morgan, A., 76-2120
Morgeli, B., 76-2157
Morishita, M., 76-1705
Moriya, M., 76-2259
Morpurgo, G., 76-2257
76-2258
Morton, D. M., 76-2051
Motuzinskiy, N. F., 76-1672
Moutschen-Dahmen, J., 76-2251
Movsesyan, N. A., 76-2343
Mueller, W., 76-1876
Mueller, W. F., 76-1894
Mukerjee, S. K., 76-1884
Mukhantsev, V. P., 76-1803
Mukherjee, S., 76-2008
Mumma, R. O., 76-2088
Mungikar, A. M., 76-1989
Munnecke, D. E., 76-1743
Murai, T., 76-2126
Murakami, M., 76-1962
Murata, H., 76-1852
Muriel, W. J., 76-2249
Muthuswamy, P., 76-1879
N'Diaye, L., 76-1768
Nadzhimutdinov, K. N., 76-1819
Nagamatsu, K., 76-2213
Nagata, H., 76-1703
Nagy, G., 76-1678, 76-1685
Nakai, K., 76-1741
Nakakoshi, R., 76-1706
Nakamura, H., 76-1856, 76-2188
76-2189, 76-2190, 76-2191, 76-2192
76-2193
Nakamura, S., 76-1648
Nakamura, T., 76-2349
Nakano, H., 76-2274
Nakatsugawa, T., 76-2287
Nalbandyan, R. A., 76-1671
Nalewaja, J. D., 76-1668
Naon, R., 76-1947
Naqvi, S. N. H., 76-1995
Nash, R. G., 76-2021
Nasilova, V. V., 76-1794
Naylor, D. V., 76-1908
Neal, R. A., 76-1728
Nearpass, D. C., 76-2156
Nehring, N. L., 76-1629
Nelson, J. H., 76-2237
Nelson, P. A., 76-2287
Nelson, P. L., 76-2318
Nene, Y. L., 76-1911
Nepoklonov, A. A., 76-1797
Neves, R. J., 76-2322
Nevzorov, M. I., 76-1848
Newsome, W. H., 76-2087
Niemczuk, R., 76-1756
Niewiadomska, A., 76-1640
Nishida, K., 76-1614
Nishijima, O., 76-2190, 76-2193
Nishikawa, T., 76-1705
Nishiyama, K., 76-1934
Nistor, C., 76-1651
Noack, G., 76-1823
Noda, H., 76-1705
Nogueira, D. P., 76-1870
Nomura, M., 76-1961
Nomura, S., 76-1707
Northcote, T. G., 76-1631
Novak, A. F., 76-1857
Nowak, A., 76-2342
Nye, D. E., 76-1722
O'Sullivan, M., 76-1844
Ochiai, F., 76-2212
Ochiai, M., 76-2152
Oe, M., 76-2081
Oe, T., 76-1912, 76-1948
Oehler, D. D., 76-2028
Oesterhelt, G., 76-1900
Ogata, H., 76-1852
Ogata, J. N., 76-1632
Ogawa, T., 76-1627, 76-1628
Ogura, K., 76-1705
Ohotomi, M., 76-2152
Okazaki, H., 76-2184
Okonek, S., 76-2201, 76-2227
Olgaard, M., 76-1783
Olkowski, H., 76-2103
Olkowski, W., 76-2103
Oloffs, P. C., 76-1631, 76-2140
Olsson, M., 76-2139
Omaye, S., 76-1712
Omiya, A., 76-1741
Ordonez, C. R., 76-1609
Orlova, N. V., 76-2246
Ortali, V., 76-2254
Orzalesi, H., 76-2321
Osborne, D. J., 76-2089
Ossa, A. P., 76-2210
Otsuka, J., 76-2274
Owen, P. J., 76-1960
Owen, W. J., 76-1737
Owens, J. C., 76-1620
Pachecka, J., 76-2268
76-2271,
Paez, D. M., 76-1830, 76-1836
Pal, R., 76-1821
Palecek, F., 76-1744
Pallotti, G., 76-1646

Palmer, J. S., 76-1621
Palusova, O., 76-2355
Palut, D., 76-1763
Pandey, S. Y., 76-1884
Pandya, K. P., 76-1996
Pantaleon, J., 76-1869
Pao, K. C. H., 76-1859
Paradowski, S, 76-1642
Pardue, J. R., 76-2079
Park, C. N., 76-1784
Parkes, D. C., 76-1958
Parmar, B. S., 76-1884
Patocka, J., 76-1972
Pavlova, N. N., 76-1943
Pawar, S. S., 76-1989
Pena Yanez, A., 76-1765
Penner, D., 76-1816, 76-1817, 76-2168 76-2334, 76-2335, 76-2336
Pennington,N. E., 76-2237
Periera, C. A. B., 76-2264
Perman, W. H., 76-1954
Persoone, G., 76-1799
Peterson, S. C., 76-1929
Petrova, E., 76-1952
Peyre, P., 76-2321
Pfister, R. M., 76-1986
Phatak, S. C., 76-1818
Phillips, G. A., 76-2137
Piacentino,H., 76-1611
Piasecki, J., 76-2178
Pickens, L. G., 76-2028
Pickering, L. K., 76-1676
Piechocka, J., 76-1843
Piedade, J. R., 76-1826, 76-2078
Piedallu, M. A., 76-2162
Pietraszek, Z., 76-1687, 76-2207
Pigati, P., 76-1867, 76-2077
Pikaar, S. A., 76-2202
Pilinskaya, M. A., 76-1786
Pineda, E. V., 76-1596
Pittman, K. A., 76-2242, 76-2243
Pitts, G., 76-2029
Plass, R., 76-2247
Plesnicar, M., 76-1716
Plimmer, J. R., 76-1690, 76-2132
Ploszynski, M., 76-1652
Poehlmann, E., 76-2223
Pol'chenko, V. I., 76-1698
Polishchuk, L. R., 76-2330
Pollero, R., 76-2122
Poloz, D. D., 76-2329
Polyak, N. P., 76-2001
Polyakov, D. K., 76-2319, 76-2320
Pomerantsev, V. I., 76-2351
Pont, V., 76-1745, 76-1814
Popa, G., 76-2211
Potapenko, N. N., 76-1806
Powell, D. M., 76-1882
Prasad, R., 76-1970
Preissig, S. H., 76-1777, 76-2044
Prestel, D., 76-1980
Preussmann, R., 76-2240
Previtali, C., 76-1902
Price, C. E., 76-2034
Prince, D. K., 76-1851
Prince, H. H., 76-1723
Prosic, Z., 76-1849
Puchkova, E. A., 76-2320
Que Hee, S. S., 76-2125, 76-2165
Quistad, G. B., 76-2025
Rabello, M. N., 76-2264
Rachev, R., 76-1664
Radomanski, T., 76-1640
Radovici, E., 76-1773
Ragab, M. T. H., 76-2065
Raghu, K., 76-1878
Raguraj, R., 76-1879, 76-1914
Raileanu, L., 76-2331
Raiskup, J. C., 76-1924
Rajukkannu, K., 76-1879, 76-1914
Raloff, J., 76-1926
Ramade, F., 76-1968
Ramasso, J., 76-1710
Rao, R. M., 76-1857
Rashad, M. N., 76-1967
Rashid, S., 76-1995
Raszewski, W., 76-1709
Ray, B. K., 76-2010
Rayner, A. C., 76-1824
Reddy, K., 76-1712
Reichert,E. L., 76-1967
Reimold, R. J., 76-1636
Reinhardt, C. F., 76-2204
Rejmanek, M., 76-1809
Rengarajan, T., 76-2348
Resnick, C., 76-1892
Restuit, A., 76-1869
Revesz, T., 76-2244
Reyes Najera, R., 76-1693
Rhodes, M. L., 76-1982
Ribas, C., 76-1867, 76-2079
Rice, C. P., 76-1828
Richard, J. J., 76-1629, 76-2094
Richou-Bac, L., 76-1869
Richter, D., 76-1940
Riedel, O., 76-1764
Rieger, H., 76-2227
Riera de Martinez, N. L., 76-1673
Rikimaru, T., 76-1706
Rissanen, K., 76-2110
Ritter, G., 76-1820
Rivera, M., 76-1841
Rivera, R., 76-1782
Riveros, M. C. K., 76-1831, 76-1832
Robbins, A., 76-1776
Roberts, D., 76-2056
Robinson, C. P., 76-2046
Robinson, E. L., 76-2170
Robinson, J., 76-1757
Rodriguez, J. G., 76-1992
Rodriguez-Kabana, R., 76-2029
Roffi, J., 76-1968
Rogers, L. J., 76-1737
Rogers, R. W., 76-1901
Romancik, V., 76-1923
Ronald, K., 76-2150
Roohi, R. A., 76-1995
Rosalki, S. B., 76-1752
Rozen, R., 76-1947
Rozo, M., 76-2158
Rubtsov, A. F., 76-2217
Rudowski, W., 76-1641
Rueppel, M. L., 76-2080
Runde, H., 76-1940
Ruveda, M. A., 76-1612
Ruzicska, P., 76-2245
Ruzo, L., 76-2018
Ryzhikov, V. D., 76-1759
Sackmauerova, M., 76-1874, 76-2135 76-2142, 76-2355
Sadler, K. C., 76-1802
Safe, S., 76-2018
Saha, J. G., 76-2037
Saidak, W. J., 76-2164
Saito, E., 76-1852
Saito, M., 76-1703
Saito, T., 76-1720
Sakamoto, T., 76-2347
Sakurai, H., 76-1837
Sakurai,T., 76-1706
Salem, H. A., 76-1903
Samek, V., 76-1858
Sanchez de la Fuente, E., 76-1693
Sandor, P., 76-2245
Santelmann, P. W., 76-1666, 76-2169
Santolucito, J. A., 76-1726
Sasaki, K., 76-2225
Sasaki, S., 76-1647
Sato, I., 76-1934
Sato, R., 76-2185
Savel'eva, M. N., 76-1788
Savina, A. S., 76-1682
Saxena, S. C., 76-2235 76-2280
Sazonov, P. V., 76-2340
Scarsbrook, C. E., 76-1624
Schanker, L. S., 76-1754
Schaper, R. A., 76-2234
Schein, L. G., 76-2053
Schewe, T., 76-1938
Schmahl, D., 76-2240
Schmid, M., 76-1760
Schmidt, G., 76-2149
Schmidt, R., 76-2279
Schmidt, S., 76-2114
Schooley, D. A., 76-2025
Schreier, K., 76-1916
Schuepbach, M., 76-2250
Schulz, K. L., 76-1750
Schwartz, L. W., 76-1713
Schwemmer, B. A., 76-1750
Schwerd, W., 76-2223
Scura, E. D., 76-2100
Seawright, A. A., 76-1957
Secreast, M., 76-1661
Sedokur, L. K., 76-2006
Segebade, C., 76-1840
Seiler, J. P., 76-2253
Selhorst, J. B., 76-1684
Selim, H. M., 76-1655
Selivanova, A. S., 76-1803
Sellers, L. G., 76-1620
Selye, H., 76-1939
Senser, F., 76-1976
Senyushkin, A. F., 76-1804
Sergeeva, D., 76-1811 76-1663, 76-1664 76-1846, 76-1847

Serrano, J. J., 76-2321
Sethunathan, N., 76-2124
Shabi, E., 76-2134
Shah, P. V., 76-1785
Sharma, M. L., 76-2277
Sharma, P. R., 76-2238
Sharma, R. P., 76-2052
Sharma, S. C., 76-2048
Sharom, M. S., 76-1667
Shealy, M. H., Jr., 76-1636
Shepelev, V. M., 76-1682
Shephard, D. A. E., 76-1680
Sheykov, N., 76-1952
Shibata,H., 76-1870
Shibazaki, T., 76-1852
Shields, J. B., 76-1781, 76-1999
Shinohara, H., 76-2152
Shirasu, Y., 76-2259
Shirosaki, E., 76-1706
Shrikhande, J. G., 76-2040, 76-2041
Shushu, G. E., 76-1659
Siddaramappa, R., 76-2124
Siddiqui, K. A. I., 76-2009
Sieck, R. F., 76-2089
Siewierski, M., 76-2115
Sikorska, M., 76-2268
76-2270
Silber, J., 76-1902
Silbera, N., 76-1902
Silk, P. J., 76-1891
Silverman, S., 76-1713
Sim, V. M., 76-1779
Simon, F. A., 76-1676
Simon, G., 76-2248
Simonffy, Z., 76-1860
Singhal, J. P., 76-1654
Sinha, S. N., 76-2010
Siquiroff, M., 76-2122
Sitkiewicz, D., 76-2269
76-2272
Skonieczna, M., 76-2270
Skryabin, G. K., 76-1990
Slizewski, M., 76-2273
Slizhevskaya, I. A., 76-1658
Smejtek, P., 76-1954
Smelt, J. H., 76-1665
Smerling, M., 76-2222
Smirnov, V. M., 76-2350
Smirnova, E. V., 76-2246
Smirnova, L. A., 76-2161
Smit, T., 76-1695
Smith, G. F., 76-1603
Smith, J. N., 76-2281
Smith, M. S., 76-1742
Smith, P. W., 76-2046
Smith, R. M., 76-2050
Smoczynski, S., 76-1653
Sodergren, A., 76-2159
Solomakha, A. I., 76-1771
Solov'eva, T. F., 76-1990
Sorsa, M., 76-2252
Soto, H. R., 76-2210
Southwick, L. M., 76-2064
Spann, J. W., 76-1801
Speir, T. W., 76-2281
Speranskaya, V. N., 76-2320
Sprankle, P., 76-1816, 76-2168
Sram, R. J., 76-1770
Staiger, L. E., 76-2025
Stamm, E., 76-2173
Steen, J. A., 76-2318
Steffens, W., 76-2015
Stein, V., 76-2242
Steinwandter, H., 76-1978
Stepanek, O., 76-1925
Stephenson, G. R., 76-1667, 76-1818
Stern, P., 76-2003
Stevanovic, M., 76-1941
Stewart, D. K. R., 76-1881, 76-2123
Stewart, R. R., 76-2287
Still, G. G., 76-2020
Stoneburner, D. L., 76-2237
Stopford, W., 76-2105
Strzelec, A., 76-1708
Sturgis, D., 76-1824
Sturino, E. E., 76-2096
Sturrock, R. F., 76-1613
Suba, L. A., 76-2080
Subramaniam, T. R., 76-1914
Suchomski, T. J., 76-2221
Suchy, M., 76-1900
Sud, S. C., 76-2007
Sugaya, A., 76-1647
Sugaya, H., 76-1701
Sulinski, A., 76-2268
76-2271
Sun, L. T., 76-1872
Sundaram, K. M. S., 76-1619
Suteu, D., 76-1951
Sutherland, R. G., 76-2125, 76-2165
Suzuki, A., 76-1648, 76-1650, 76-1933
Suzuki, H., 76-1705, 76-2212
76-2274
Suzuki,K., 76-1703, 76-2187
76-2274, 76-2346
Suzuki, T., 76-1701, 76-1730
Suzuki,Y., 76-1647, 76-1961, 76-2345
Svec, H. J., 76-1629, 76-2094
Svobodova, Z., 76-1973
Swim, H. E., 76-2016
Syrowatka, T., 76-1763
Szeto, S. Y., 76-1631
Szilagyi, M., 76-1678
Szokolay, A., 76-1874, 76-2135
Szumilak, G., 76-1652
Tabata, K., 76-1720
Takacs, J., 76-1860
Takahashi, K., 76-1703
Takamatsu, M., 76-1706
Takamiya, T., 76-1649, 76-1704
Takano, S., 76-1703
Takaoka, Y., 76-1837
Takats, G., 76-2205
Takayama, C., 76-1648
Takeda, M., 76-1730
Takeuchi, T., 76-1706
Takiura, K., 76-2081
Tamagawa, S., 76-2183
Tan, B.J., 76-2281
Tanaka, K., 76-1733
Tang, H. V., 76-1746
Tashkhodzhaev, P. I., 76-1819
Tassara, R. L., 76-1711
Tauris, P., 76-1697
Taylor, A. W., 76-1899
Taylor, H. W., 76-2239
Taylor, J. R., 76-1684, 76-2045
Teh-fang, V., 76-2179
Teleugaliev, T. M., 76-2160
Tengan, L. S., 76-1632
Terekhov, A. V., 76-1769
Ternovoy, V. I., 76-1789
Teruya, H., 76-1706
Tevan, L., 76-1991
Thielemann, H., 76-2175
Thirugnanam, M., 76-1998
Thirumurthi, S., 76-1914
Thomas, J. A., 76-2053
Thomas, R., 76-1745
Thomason, I. J., 76-1887
Thompson, C. K., 76-2356
Thomson, J. M., 76-2154
Thullen, R. J., 76-2167
Thurlbeck, S. M., 76-1927
Thurlbeck, W. M., 76-1927
Thurzo, A., 76-1678
Thurzo, T., 76-1685
Tishkov, A. I., 76-2328
Titov, V. V., 76-1806
Titova, G. S., 76-1806
Tkach, L. I., 76-1698
Todorov, D., 76-1663, 76-1811
Todorova, M., 76-1847
Tokoro, T., 76-2274
Tolle, A., 76-1886, 76-2102
Tollefson, J. J., 76-1620
Tomaru, K., 76-2345
Tomatis, L., 76-1870
Tomio, T., 76-1707, 76-2275
Tomizawa, C., 76-2126, 76-2187
Tomov, A., 76-1601
Tong, C., 76-2321
Topchyan, A. A., 76-2343
Torchinsky, A. M., 76-2246
Toro Rojas, F., 76-1765
Torres Velasco, F., 76-1765
Tourte, J., 76-1845, 76-1863, 76-2072
Touzeau, J., 76-1600
Toyama, T., 76-2183
Traczyk, Z., 76-1641, 76-1643
Trautmann, K. H., 76-1900
Treble, D. H., 76-2243
Trenel, J., 76-2216
Trubowitz, S., 76-1935
Truchlinski, J., 76-2057, 76-2058
Truhaut, R., 76-1983
Truong, H. M., 76-1746
Tsuge, S., 76-2187, 76-2346
Tsukano, Y., 76-2186
Tsunenari, S., 76-2083
Tsutsui, J., 76-1705
Tsvetkova, T., 76-1847
Tumino, R., 76-2122
Tyagi, M. P., 76-1734
Uamagishi, M., 76-1703
Uchida, A., 76-1649, 76-1704
Uchiyama, M., 76-1730, 76-1740
76-1961, 76-2185

Ueki, S., 76-1702, 76-2225
Uhnak, J., 76-1874, 76-2135, 76-2142
Ungaro, M. T., 76-2264
Unger, I., 76-1891
Unterhalt, B., 76-1625
Unterman, H. W., 76-1651
Ushio, F., 76-1614
Usutani, S., 76-1934
Uthe, J. F., 76-2150
Uzakov, U. Ya., 76-2319, 76-2320
Vainio, H., 76-1718
Valange, B., 76-1838
Valencia, R. A., 76-2229
Valjevac, K., 76-2003
Van T Laar,A., 76-1683
Van Alfen, N. K., 76-2019
Van Assche, C., 76-1623
Van de Veire, R., 76-2042
Van den Bosch, R., 76-2103
Van Gelder, G. A., 76-2050
Van Hees, P. A. M., 76-1683
Van Heijst, A. N. P., 76-2202
Van Peteghem, C. H., 76-2091
Van Ulsen, F. W., 76-1696
Van Wambeke, E., 76-1623
Vanachter, A., 76-1623
Vance, B. D., 76-2236
VanMiddelem, C. H., 76-1630
Vanwetswinkel, G., 76-1597
Vaquer, A., 76-1657
Varga, G., 76-1678, 76-1685
Varty, I. W., 76-1616
Vasavada, P. C., 76-2033
Vasques,R. M. P., 76-1826, 76-2078
Vasyukova, E. A., 76-1759
Velcich, A., 76-2257, 76-2258
Venkitasubramanian,T. A., 76-2048
Verlaat, J. G., 76-1665
Verma, S. R., 76-1734
Vijan, P. N., 76-1824
Virgo, B. B., 76-1997
Viscido, A. A., 76-1829
Visweswariah, K., 76-2061
Vizek, M., 76-1744
Vodden, H. A., 76-1762
Vodyanov, A. A., 76-1796
Volk, V. V., 76-2172
Volynchuk, I. M., 76-2324
Von Stryk, F. G., 76-2164
Vonesch, E. E., 76-1609, 76-1831 76-1832
Vonk, J. W., 76-2036
Vucemilo, M., 76-1810
Vyas, S. C., 76-1911
Wachendorff, R., 76-2215
Wada, K., 76-1705
Walker, A., 76-2133
Walker, C. H., 76-2282
Walker, W. W., 76-1909
Wallnoefer, P., 76-1977
Ward, D. V., 76-1993
Wardowski, W. F., 76-1890
Washburn, R. H., 76-1621
Waskey, C. E., 76-1903
Wassermann, D., 76-1870
Wassermann, M., 76-1870
Watabe, K., 76-1647
Watanabe, C., 76-2190, 76-2191
Watanabe, P. G., 76-2052
Watkins, D. A. M., 76-1875
Weber, H., 76-1747
Weeraratna, C. S., 76-1742
Wegman, R.C.C., 76-2144
Weinberger, P., 76-1970
Weisgerber, I., 76-1980, 76-2035 76-2109
Weissmuller, J., 76-1964
Wellenstein, G., 76-1645
Wells, M. R., 76-1960
Wendel, K. G., 76-2357
Wendlandt, E., 76-2176
Westoo, G., 76-1662
Wheeler, W. B., 76-1630, 76-2132
Whitacre, D. M., 76-1750
Whitcomb, E. R., 76-1726
White, D. H., 76-1634, 76-1637
White-Stevens, R. H., 76-2128
Whitehead, P. H., 76-2266
Whitten, C. J., 76-2022
Wiedmann, J. L., 76-2020
Wieneke, J., 76-2015
Wiener, M., 76-2242, 76-2243
Wierzbicka, M., 76-1748, 76-1749
Williams, B., 76-2079
Williams, I. H., 76-1896
Willis, G. H., 76-2064
Wilms, H., 76-2173
Wilson, A. J., 76-2344
Wilson, B. B., 76-1903
Wilson, D. J., 76-1861
Winnett, G., 76-2115, 76-2128
Wipf, H.K., 76-1900
Wittenberger,C., 76-1951
Wojcik, D. P., 76-1630
Wolfe, L. S., 76-1739
Wolfe, N. L., 76-1725
Wolff, F., 76-1699
Wolff, F. C., 76-2210
Wolfe, J., 76-1919
Wolring, G. Z., 76-1988
Wood, G. R., 76-1824
Wood, G. W., 76-2123
Woodard, B. T., 76-1861
Worth, H. M., 76-1780
Wright, D. J., 76-1986
Wurster, C. F., 76-1675
Wybieralska, A., 76-2178
Wybieralski, J., 76-2177, 76-2178
Wyllie, J. A., 76-1633
Yamaji, A., 76-2081
Yamamoto, H., 76-2276
Yamamoto, S., 76-1705
Yamashita, S., 76-2193
Yamashita, Y., 76-2345
Yanagisawa, T., 76-1702, 76-2225
Yang, R. S. H., 76-1894
Yarbrough, J. D., 76-2286
Yaron, B., 76-1897
Yaroshenko, V. I., 76-2354
Yoder, J., 76-1633
Yokoyama, T., 76-1648, 76-1702
Yoneda, H., 76-1826, 76-2078
Yonova, I., 76-1812
Yoshida, T., 76-1961
Yotov, Y., 76-1663, 76-1664, 76-1811
Yuki, H., 76-2081
Yule, W. N., 76-1616
Yurukov, M., 76-1811
Zalewska, Z., 76-2269 76-2272,
Zamora, M. M. K., 76-1829
Zandvoort, R., 76-1665
Zatula, A. I., 76-1848
Zelinskiy, M. I., 76-2323
Zepp, R. G., 76-1725
Zerba, E. N., 76-1835
Zhecheva, G., 76-1812
Zherebchenko, P. G., 76-1769
Zhilenko, V. N., 76-2005
Zhorov, V. O., 76-2112
Zhulenko, V. N., 76-2161
Zimmering, S., 76-2228
Ziolkowska, G., 76-2271
Znamenskiy, V. V., 76-1769
Zonderwijk, P., 76-1607
Zufarov, K. A., 76-1819
Zurawski, H., 76-1652
Zvorykina, L. B., 76-1672

Corporate Author Index

Counc. Agric. Sci. Technol. 76-2113

Hyg. Lab., Okayama Prefect. Govt. 76-2194

Nagasaki Prefect. Off. 76-2180

Saga Prefect. Off. 76-2195

The Administrator of the U.S. Environmental Protection Agency has determined that the publication of this periodical is necessary in the transaction of public business required by law of this Agency. Use of funds for printing this periodical approved by the Director of the Office of Management and Budget February 6, 1973.

Pesticides Abstracts is available on 16 mm microfilm. Additional information can be obtained from the Technical Services Division.

ORDER FORM FOR
PESTICIDES ABSTRACTS

To: Superintendent of Documents
Government Printing Office
Washington, D.C. 20402

Please enter my subscription for Pesticides Abstracts. I am enclosing Money Order ☐ Check ☐ for this subscription.
($18.25 a year; $4.60 additional for foreign mailing.)

Please address as follows: _____

_____

_____

UNITED STATES GOVERNMENT PRINTING OFFICE, WASHINGTON, D.C. 1968

For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402. Subscription price, domestic, $18.25 a year, $4.60 additional for foreign mailing. Price for a single copy of this issue is $1.40, Index issue $2.35.

U.S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF PESTICIDE PROGRAMS
TECHNICAL SERVICES DIVISION
RM. EB-45, 401 M STREET, S.W.
WASHINGTON, D.C. 20460

POSTAGE AND FEES PAID
FOURTH CLASS RATE BOOK
EPA-335

U.S.MAIL

Serials Department
Xerox University Microfilms
300 North Zeeb Road
Ann Arbor, MI 48106 EPA 18-1

MISC.

If you do not desire to continue receiving this publication, please CHECK HERE ☐ ; cut off this label and return it to the above address. Your name will then be removed from the mailing list.